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ANNALS
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VOL. 44. MARCH, 1935. No. 1.

I.

THE CELLULAR CHARACTER OF ONE HUNDRED TEMPORAL BONES: CLINICAL AND SURGICAL SIGNIFICANCE.

EDWARD F. ZIEGELMAN, M. D.,

SAN FRANCISCO.

The development of the temporal bone from three different parts: the pars petrosa, the pars squamosa and the tympanic ring, demands three separate cell systems. This is of importance when referable to its diagnostic and surgical application.[1] In the development of bone a differentiation into two types takes place: the membranous and the cartilaginous. From the former is developed the flat bones, such as those of the cranial vault. From the latter the long bones, comprising the chief skeletal structure. When making observations on the temporal bone, it is necessary to keep this in mind, since it is a composite structure composed of a long and a flat bone. This gives it an individualism of its own.

Flat bones, such as the squamosal portion, are composed of an inner and an outer layer of compact bone. They are commonly called tables, between which is normally placed diploe. It corresponds to the cancellous type, containing marrow within its interstices. This diploe is normally of the red type, though age and disease modify it. The structure of a long bone is different. It is composed of a shaft, or diaphysis, and an epiphysis, or extremity. Between the diaphysis and

(It is with a sense of deep regret that the ANNALS announces the death of Doctor Edward F. Ziegelman, author of this article, on October 12, 1934. His untimely loss deprives this journal of one of its most valued contributors.)

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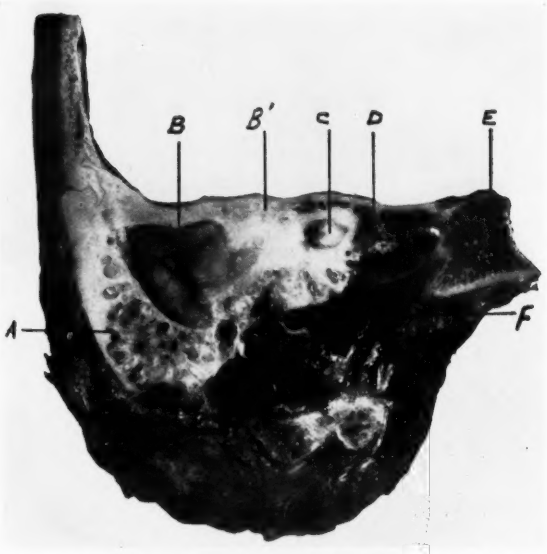

Fig. 1. Photograph of a section through the posterior plane of a temporal bone in situ. It shows at *A* a diplopneumatic mastoid; *B* the posterior fossa of the skull; *B'* diploetic cells extending into dark diploetic tip at *D*; *C* the internal auditory meatus; *E* the sphenoid base; *F* the jugular fossa. Notice how the cells extend along the inferior part of the temporal bone to ultimately reach the petrosal tip at *D*.

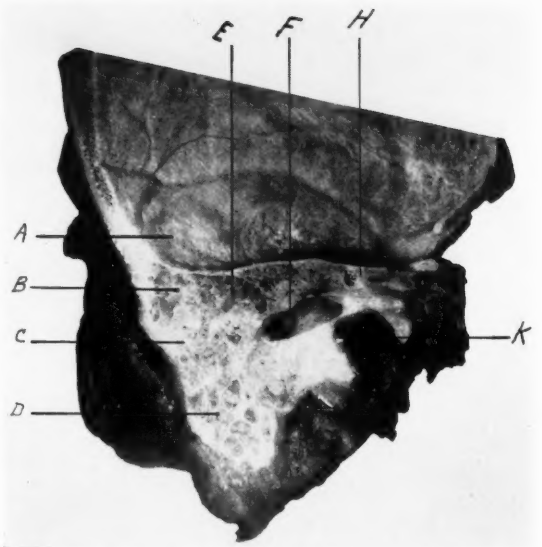

Fig. 2. Photograph of a section through a posterior plane of a temporal bone in situ. It shows at *A* the posterior fossa; *B* diploetic cells of mastoid; *C* a sclerotic area of mastoid; *D* a diplopneumatic area of mastoid; *E* a diploetic cellular area extending into a diploetic tip at *H*, *F* the internal auditory meatus sectioned at an angle; *K* the jugular fossa. From the terminals of lines *E* and *H* the structure is diploetic in this plane with the exception of a small area external to the terminal of line *H*. This area can be seen and recognized by the presence of a small air bubble. Note the marked sclerosis in this specimen just external to the jugular fossa. The black area anterior to lines *A*, *B*, *C*, *D* is the ear retracted forward.

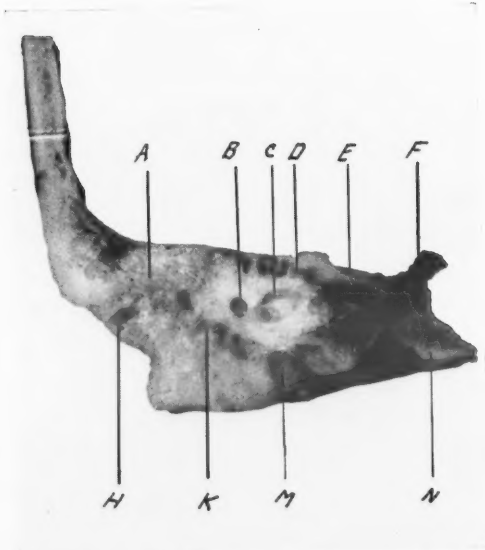

Fig. 3. Photograph of a section through a posterior plane of a temporal bone. It shows at *A* a diploesclerotic area of the mastoid; *B* a portion of the labyrinth; *C* the internal auditory meatus; *D* a diplopneumatic area extending into a diploetic tip; *F* a part of the posterior clinoid process; *H* a pneumatic cell; *K* diplopneumatic cells extending along inferior margin of otic capsule to point *M* a part of the jugular fossa; *N* the sphenoid base. In this specimen a portion of the mastoid has been removed as represented by area in proximity to terminal of line *H*. Also note how the cells extend from area *A* along upper margin to petrosal tip as represented by letter *E*.

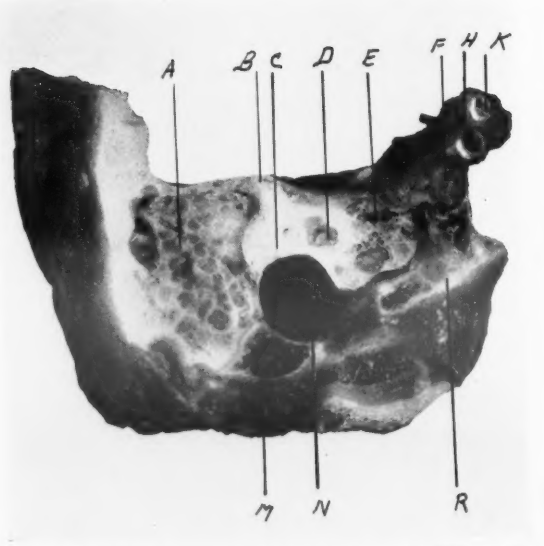

Fig. 4. Photograph of a section of a temporal bone in a posterior plane. It shows at *A* a dissected diplopneumatic mastoid; *B* cells extending along superior margin to diplopneumatic tip at *E*; *C* otic capsule; *D* internal auditory meatus; *F* posterior root of gasserian ganglion; *H* and *K* section through internal carotid lateral to sella. The structures represented by *F*, *H* and *K* have been sectioned on a more anterior plane and separately to show them; *M* a part of occipital bone; *N* the jugular fossa; *R* the sphenoid base. Dura still adherent to upper surface of petrosal tip.

epiphysis is placed the epiphyseal line, from which much of the bony growth takes place. A cross section demonstrates an outer compact layer, within which is a cancellous structure and marrow cavity, usually yellow in color. Though the long bones have a central marrow cavity, the epiphysis and in cancellous bone generally, the diploe persists.[2,3] The petrosal pyramid is such a bone. It has a shaft: the pyramid, and an apex or extremity: the epiphysis. As in other long bones, the diploe is originally present within the cancellous bone of the shaft, as well as in the apex or epiphysis. Being closely associated with the mucous membrane of the middle ear and mastoid antrum, the normal tendency is for the substance of the diploe to be absorbed and converted by invagination of this mucous membrane into a pneumatic cellular character, unless early infection modifies this invasion.[1] Recent work seems to show that other factors are concerned as well.[5] Though the writer is in hearty accord with Wittmaack, observations seem to reveal that certain individuals are more resistant to this invasion. To be more specific, some are more diploetic than others.

In this presentation of the cellular appearance of a series of temporal bones, an attempt has been made to present the subject from the aspect of a structure composed of compact osseous walls, within which was originally diploetic tissue. By diploetic tissue, I infer marrow within a cancellous bony structure. It harbors little of the microscopic, is based upon gross observations (Figs. 1-19), and endeavors to present a mental picture that will be of value to the clinician and surgeon. Consequently the otic capsule, its development and ossification assumes no place in this presentation. Investigations of this particular area of the temporal bone have been and are still being carried out. I refer to the work of Fraser, Bast and others.[6-9]

A certain basic classification has been necessary to clarify the observations. It differs little from that used by other observers, whether based upon microscopic or macroscopic findings.[10-12] It is one of gross cellular appearance relative to a definite anatomic location. All observations were made by the writer, most of them from cadaver specimens, though a limited number were from fresh autopsy material. Included in the 100 specimens studied were three which demonstrated gross pathologic lesions in the middle ear, manifested by partial or complete loss of the drum, with loss of part or all of the incus and malleus. Special note was made of these. The specimens were obtained at random and but few of the clinical histories were known. The temporal bones were sectioned either in the vertical, longitudinal or horizontal plane of the pyramid and apex at approximately four mm. distances. From four to seven sections were made

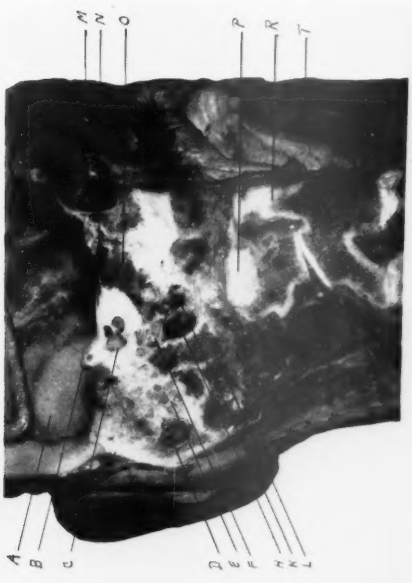

Fig. 6. Photograph of a section through the temporal bone and related structures. It shows at *A* the posterior fossa; *B* the superior semicircular canal; *C* the labyrinth with part of vestibule and cochlea; *D* the petrous part of temporal; *E* pneumatic area of mastoid; *F* a diploemmatic area of both area extending to a diploetic tip at *G*; *H* jugular bulb; *K* carotid artery; *L* part of occipital bone; *M* sphenoid sinus; *N* diploemmatic cells extending from mastoid to tip on the superior surface; *O* the atlas; *P* the odontoid process of the axis; *R* the oral cavity. The specimen shows very nicely how the cells extend from the mastoid to the styloid tip.

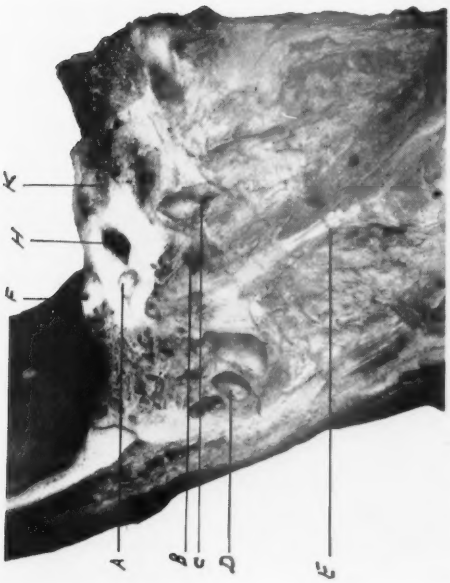

Fig. 5. Photograph of section through temporal bone and related structures. It shows at *A* the sigmoid sinus; *B* the superior semicircular canal; *C* the carotid canal; *D* a pneumatic cell; *E* the petrous; *F* the superior semicircular canal; *H* the internal auditory canal; *K* a diploetic tip. A direct communication from the diploemmatic mastoid to the diploetic tip can be seen passing over the superior semicircular canal. Notice the styloid muscles attached to the styloid tip.

from each specimen, depending upon its size. In the vertical longitudinal, the anterior section passed through the anterior portion of the middle ear, the second through the midline of the middle ear, the third in the plane of the descending portion of the facial canal, the fourth through the antrum and the others through the mastoid process, keeping in the plane of the pyramid. In addition to the gross cellular study of the respective segments, a complete dissection was made of the parts when it seemed as though added information might be obtainable. At times an eight diopter binocular loupe was used to check the findings.

Following are the observations relative to the cellular character and anatomic location of 100 temporal bones, sectioned according to the method as described in the text. The terms anterior, inferior and posterior, are used relative to the three surfaces of the pyramid. The letters d, p and s, used under the title mixed, refer to the predominant type of cell present, when classified under that of the mixed type. Chart 1 represents the observations in the ninety-seven specimens with no gross evidence of pathology in the middle ear. Chart 2, of the three specimens with gross evidence of pathology in the middle ear, and Chart 3 is a composite summary of the findings of the total number of 100 specimens studied. In Chart 3 those of the mixed type have been segregated and placed under the caption that represents the predominant type of cell found to be present.

The observations in reference to the mastoid coincide closely with those of Zuckerkandl. His findings were based upon 250 temporal bones, dry and macerated.[10] The character of the tip is similar to the observations of Yamashita,[11] in so far as pneumatization is concerned, but differs markedly from that of Balin and Balinoff.[12] It agrees with them as to other portions of the temporal bone. The small number of diploetic mastoids differs from that of Cheatle,[13] if one classifies this type exclusively as the infantile. He included in his observations the sclerotic. Applying a similar classification to the observations as presented, little difference is noted. The mixed type of cellular character, which includes over fifty per cent of temporal bones studied, showed a predominance of the diplopneumatic type. One is impressed with the small number of sclerotic bones observed. It is the writer's opinion that the predominant diploetic character of the tip may be explained on the basis that it is comparable to the epiphyseal end of a long bone. The findings as presented impress one with the fact that the cellular anatomy must be given due consideration in drawing clinical conclusions and advising therapeutic measures in infections of this structure. To assume that it is definitely diploetic,

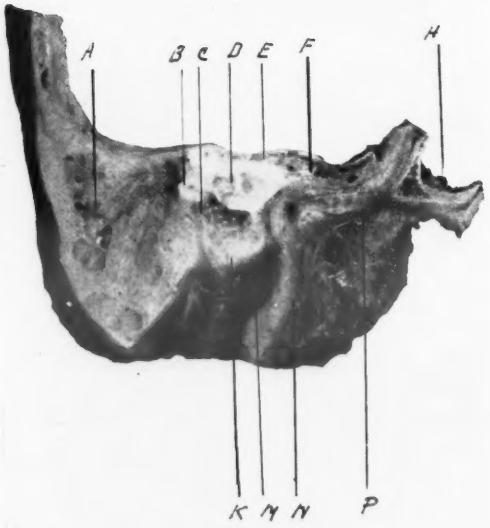

Fig. 7. Photograph of a section through the plane of the facial nerve. It shows at *A* pneumatic cells in a predominently sclerotic mastoid; *B* external semicircular canal; *C* facial canal with nerve in situ; *D* labyrinth with cochlea; *E* diplocitic cells along superior surface communicating with a diplopneumatic tip at *F*; *H* sphenoid sinus; *K* the bulb cells; M the internal jugular vein; *N* the internal carotid artery; *P* the wing of the sphenoid bone. Notice the diplocetic cells extending from the mastoid behind the facial canal to the bulb cells and direct to the carotid canal. The opening of the superior semicircular canal can be seen between the lines *C* and *D*.

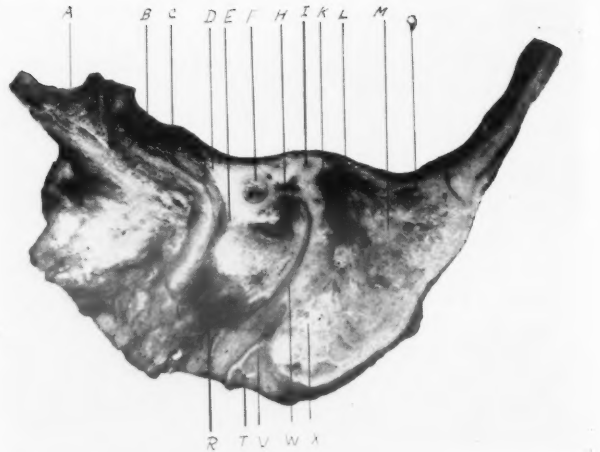

Fig. 8. Photograph of a section through the facial canal. It shows at *A* the sphenoid sinus; *B* a diplocitic tip; *C* internal carotid artery in bony canal; *D* diplocitic cells in external aspect of tip; *E* diplocitic cells extending into carotid canal from bulb area; *F* part of cochlea; *H* middle ear; *I* superior semicircular canal; *K* external semicircular canal; *L* air space extending through anterior posterior diameter of specimen the result of a dissection of the part; *M* diplocitic area; *O* pneumatic cells; *R* bulb area; *V* the occipital artery with stylo mastoid branch at *T*; *W* the facial canal with facial nerve in situ; *X* diplopneumatic area of mastoid. Notice the difference in diameter of the facial canal in the upper and lower part; also the wing of the sphenoid as it extends externally on a plane inferior to the carotid artery.

CHART I.

GROSS PATHOLOGY ABSENT IN MIDDLE EAR.

No. Temporal Bones	*Anatomic Location*	*Cellular Character*			
		Diploetic	*Pneumatic*	*Sclerotic*	*Mixed*
97	Mastoid	9	28	5	55 d-11 p-31 s-13
	Zygoma	17	21	7	52 d-21 p-19 s-12
	Pyramid				
	a—anterior	19	17	5	56 d-22 p-29 s-5
	b—inferior	22	14	8	53 d-24 p-17 s-12
	c—posterior	16	22	6	53 d-17 p-31 s-5
	Tip	72	6	1	18 d-12 p-6 s-0
	Bulb Cells	32	14	12	39 d-20 p-14 s-5

pneumatic or sclerotic in any one or all of its parts and to base one's clinical and therapeutic opinion on such a standard may be fraught with evil consequences (Figs. 1-19).

The chief interest during the past two decades in infections of the temporal bone has been its relation to the petrosal apex. This may be said to date from the presentation of a symptom complex by Gradenigo. His original decription was that of clinical evidence characterized by the presence of homolateral abducens paralysis, eye pain and facial neuralgia, in the presence of acute otitis with or without mastoid involvement.[14] Innumerable observers have made similar contributions to the literature.[15-21] Many do not agree that it rep-

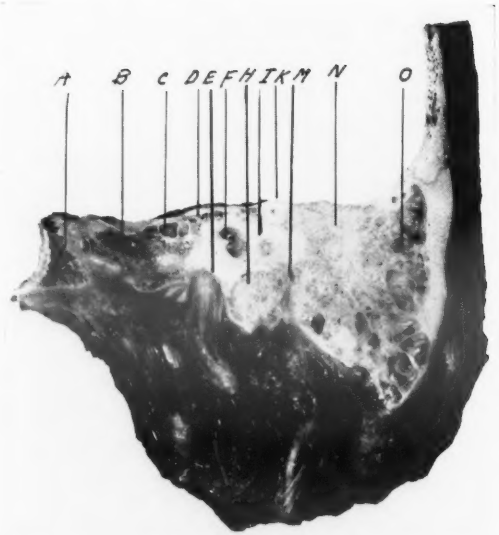

Fig. 9. Photograph of a section through the facial canal. It shows at *A* the sphenoid body; *B* a diploetic tip; *C* pneumatic cells in the diplopneumatic tip; *D* cells of communication from the mastoid to tip; *E* the internal carotid; *F* the cochlea; *H* the bulb area diploetic in type; *I* the vestibule; *K* the superior semicircular canal; *M* the facial nerve in the facial canal; *N* a sclerotic part of the mastoid; *O* pneumatic cells of the mastoid. Between *N* and *O* is a diploetic area. Notice how the diploetic area external to the facial canal communicates with the diploetic area of the bulbar cells and directly into carotid canal. The dark area below the bulb cells is the internal jugular vein. Also notice a few large pneumatic cells in the tip area just external to the diploetic tip.

Fig. 10. Photograph of a horizontal section through a temporal bone at the level of the attic. *A* diplopneumatic mastoid; *B* sigmoid sinus; *C* posterior fossa; *D* internal acoustic meatus with 7th, 8th and intermedius nerve. The nerves may be seen as they enter the internal acoustic foramen; *E* diplopneumatic zygoma; *F* diplopneumatic cells extending from zygoma to petrosal tip at *N* and *O*; *H* the head of the malleus; *I* the middle ear from above; *M* cochlea, external and posterior to it the vestibule; *K* the anterior petrosquamosal suture line; *N* and *O* diplopneumatic tip. Notice the relatively short distance from area between terminals of lines *H* and *I* to the cells at *K*. This photograph shows the cells of communication from the zygoma to the petrosal pyramid and tip.

CHART II.

GROSS PATHOLOGY IN MIDDLE EAR.

No. Temporal Bones	*Anatomic Location*	*Cellular Character*			
		Diploetic	*Pneumatic*	*Sclerotic*	*Mixed*
3	Mastoid	0	0	0	3 d-1 p-0 s-2
	Zygoma	1	0	0	2 d-1 p-0 s-1
	Pyramid				
	a—anterior	2	0	0	1 d-1 p-0 s-0
	b—inferior	2	0	0	1 d-0 p-1 s-0
	c—posterior	2	0	0	1 d-0 p-0 s-1
	Tip	3	0	0	0
	Bulb Cells	1	0	0	2 d-0 p-0 s-2

sents a definite disease entity, but that it is a symptom complex which may be present in many other pathologic lesions.[22-26] As Eagleton[27] so well states: "Symptoms of facial pain with abducens paralysis must be analyzed from the standpoint of caries of the petrosal tip, localized pontile cistern meningitis the result of such caries, suppuration of the sphenoid sinus, thrombophlebitis of the cavernous sinus and associated petrosal and basilar veins." The writer would paraphrase the above statement as follows: Any pathologic process, whether traumatic, inflammatory or of the new growth type, of or in the immediate vicinity of the petrosal tip must be considered as a possible etiologic factor. Mosher's opinion[28] that the gasserian ganglion may at times be the first structure to be invaded from adjacent or distant toxic or septic foci, presents a new and fertile field for further investigation relative to pathology in the petrosal apex and contiguous structures.

Fig. 12. Photograph of a section through a plane just posterior to the external auditory canal. It shows the tube, that is between tube and internal carotid artery. It shows at A cells of the zygoma extending to the tip B and seen just superior to the probe which lies in the anterior part of middle ear and external auditory canal. C middle loss. Note middle meningeal artery; D wing of sphenoid, it shows pneumatization; E internal carotid artery in cavernous sinus; F pituitary gland; H spheroid sinus; K pneumatic cyst in zygoma; M external auditory canal; N cheana; O posterior margin of septum; R probe in nasopharyngeal opening of eustachian tube. Note close relation of cellular contents of petrosal tip to inner middle ear wall.

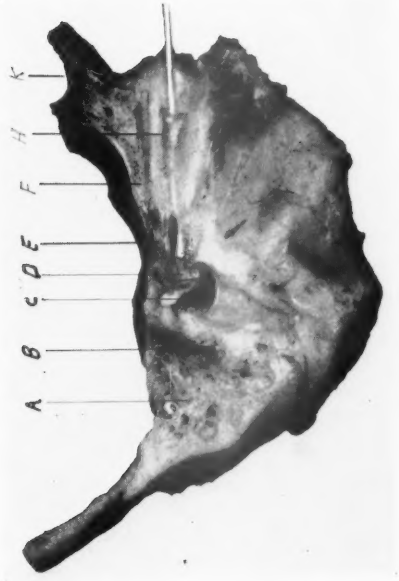

Fig. 11. Photograph of a section through a plane just anterior to the eustachian tube. It shows at A diploemmatic mastoid with marked sclerosis; B air cells at tip; C middle ear with ossicles intact, the malleus can be seen through D point of middle ear approach for localized pus in tip E. Note readily seen F wing of sphenoid often misses close proximity of petrosal pyramid. This structure is diploemmatic in this taken for H eustachian tube with probe in it; K the sella. Notice the diploetic character of cells communicating in roof of middle ear.

CHART III.

SUMMARY OF FINDINGS IN 100 SPECIMENS.

No. Temporal Bones	*Anatomic Location*		*Cellular Character—Diplocitic*	*Pneumatic*	*Sclerotic*
100	Mastoid		9	28	5
			12 mixed	31 mixed	15 mixed
		Total	21	59	20
	Zygoma		18	21	7
			22 mixed	19 mixed	13 mixed
		Total	40	40	20
	Pyramid				
	a—anterior		21	17	5
			23 mixed	29 mixed	5 mixed
		Total	44	46	10
	b—inferior		24	14	8
			24 mixed	18 mixed	12 mixed
		Total	48	32	20
	c—posterior		18	22	6
			17 mixed	31 mixed	6 mixed
		Total	35	53	12
	Tip		75	6	1
			12 mixed	6 mixed	0 mixed
		Total	87	12	1
	Bulb Cells		33	14	12
			20 mixed	14 mixed	7 mixed
		Total	53	28	19

When occurring as a result of infections in the middle ear, it extends by continuity of tissue or vascular transmission. When extension does occur by way of the pyramid, it is evident that the prevalent route would be by way of a diplopneumatic structure. This may account for the presence of an empyematous lesion with an associated osteomyelitis. When invasion of the tip occurs, it would appear that the lesion would be one of a suppurative process, with a tendency to develop into that of an empyematous type, similar to the socalled Brodie abscess familiar to the general surgeon.[29] This explains the apparent prevalence of clinical evidence of localized lesions in the tip. It is the writer's opinion that the most frequent source of such pathology is

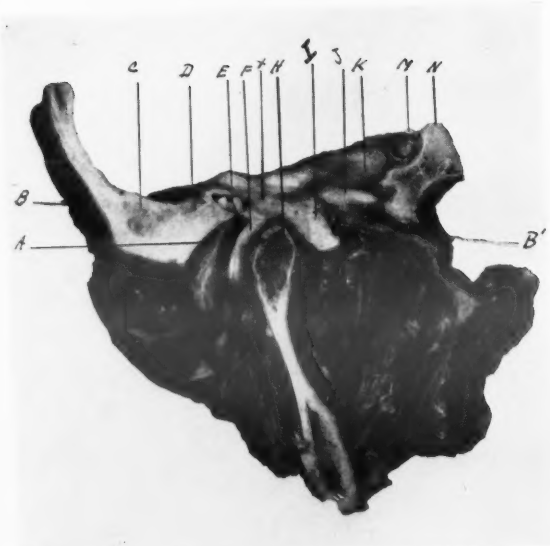

Fig. 13. Photograph of a section through a plane anterior to the eustachian tube and through the temporomandibular joint. It shows at *A* external auditory canal; *B* and *B'* probe in eustachian tube; *C* diplopneumatic zygoma with cells extending over roof of middle ear to tip at *N*; *D* cells extending from zygoma; *E* middle ear with ossicles intact; *F* vaginal process of temporal bone showing the part it takes in the formation of the temporomandibular joint; *H* condyle, neck and part of ramus of inferior maxillary bone; *K* petrosal tip; *M* cavernous sinus; *N* sphenoid bone. The lower portion of line *E* passes through the eminentia arcuata. Between line *F* and *H* is the subarcuate fossa. *I* is the anterior part of the glenoid fossa; *J* part of sphenoid wing.

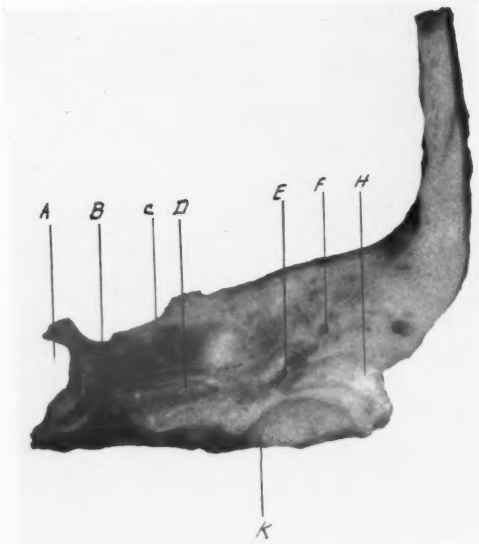

Fig. 14. Photograph of a section through the plane of the internal carotid artery. It shows at *A* sphenoid sinus; *B* sphenoid bone; *C* petrosal pyramid tip; *D* internal carotid artery opened in longitudinal plane; *E* middle ear with long handle of malleus and drum membrane intact; note the acute angle the drum membrane makes with the external auditory canal floor; *F* pneumatic cell in a sclerotic zygoma which has a fair amount of diplopneumatic cells present; *H* the external bony auditory canal; *K* the glenoid fossa.

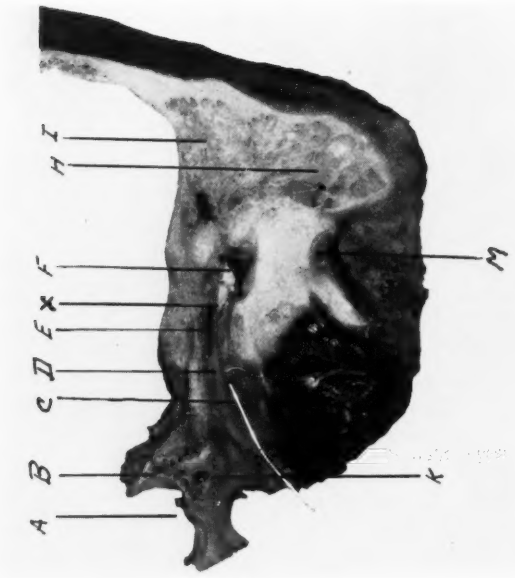

Fig. 16. Photograph of a section anterior to the eustachian tube. It shows at *A* sphenoid sinus; *B* sphenoid bone with air cells; *C* probe in eustachian tube; *D* sphenoid wing; *E* petrosal tip; notice its close relation to middle ear *F*; *H* diploepneumatic cells of mastoid; *I* diploetic cells of mastoid; *K* pterygoid canal; *M* glenoid fossa. It is at point *X* where approach is made from middle ear to reach localized pus in the tip.

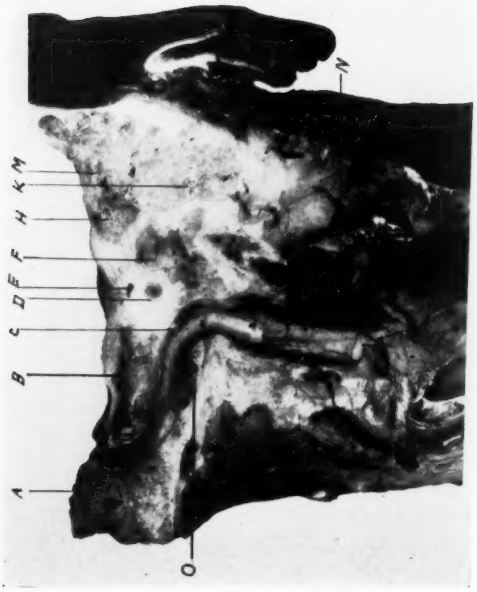

Fig. 15. Photograph of a section through the plane of the middle ear. This specimen had a chronic purulent otitis media. The malleus and incus were destroyed. It shows at *A* the sphenoid base; *B* petrosal tip diploetic in type; *C* the carotid artery and canal; *D* part of the cochlea; *E* great superficial petrosal nerve; *F* middle ear; notice sclerosis about it except at lower part or floor where diploetic cells extend into carotid canal; *H* diploetic cells of mastoid; *K* pneumatic cells of mastoid; notice air bubble; *M* diploepneumatic area of mastoid; *N* internal jugular vein; *O* sphenoid wing; notice pneumatization in it. This specimen demonstrates clearly how an extension from the middle ear may involve the carotid canal with subsequent involvement of the tip.

from the middle ear and mastoid, and that it may extend by any one of several planes.

The surgical anatomy as presented favors the posterior aspect of the pyramid as being the most prevalent plane of invasion (Figs. 1-6), then from the zygoma directly (Figs. 10-13), or from the middle ear by way of the peritubal cells or carotid canal (Figs. 11, 13-16), and finally from the bulbar or retrofacial cells (Figs. 7-9, 17, 18). The complexity and variability of the cellular character of the temporal bone compels one to accept the trite and euphonious statement that each one is a law unto itself. This applies to cellular invasion, diagnosis and therapy. It agrees with the opinion of others who conclude that there is no one procedure capable of handling infections of all temporal bones.[30] The term petrositis, made popular by Profant,[17] was not intended by him to designate the type of pathology present. It is indicative of a general inflammatory process. It possesses the same value that femuritis or humeritis would have when applied to an inflammatory lesion of the femur or humerus. When, and if used, it should be qualified by the specific pathologic term that represents the pathology present. In temporal bones a periostitis, osteitis, osteomyelitis or suppurative process, with or without abscess formation, may occur, either independently or in combination. These pathologic processes may assume innumerable terminal forms, chief of which may be mentioned rarefaction, sclerosis, necrosis, caries and sequestration. The final result depends upon the cellular character of the temporal bone, the resistance of the individual, the part and type involved, the method of invasion, the character of the infection present and the type of therapy used. We must go back to our surgical anatomy and surgical pathology applicable to bony structures, to obtain a clear conception of the lesions that may occur in the temporal bone and apply the principles of general surgery to the therapy of such pathology.

When the tip is involved and the pathology is one of the exudative type, purulent or empyematous in character, observations definitely indicate that the ideal method of approach is by way of the peritubal cells.[31] This surgical conclusion is based upon the surgical anatomic fact that the lateral aspect of the tip or, as I prefer to call it, the epiphysis, is very close to the anterior aspect of the inner wall of the tympanic cavity, making it the shortest route of approach to the tip (Figs. 10-13, 16). The structure to avoid is the internal carotid artery. It is posterior to the peritubal cells (Figs. 11-15, 17, 19).

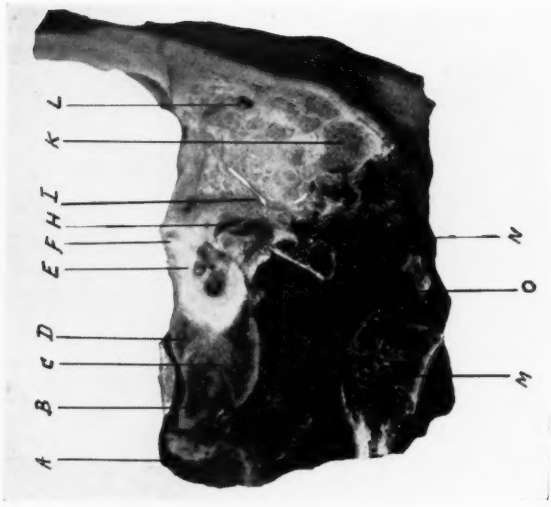

Fig. 18. Photograph of a section through facial canal. It shows at *A* spheroid sinus; *B* diploetic tip; *C* part of sphenoid wing; *D* diploemmatic cells in external part of petrosal tip; *E* the labyrinth showing cochlea and vestibule; *F* superior semicircular canal; *H* facial canal opened; *I* orifice of facial canal; *K* diploemmatic cells to facial nerve; *L* probe posterior to facial canal; *M* diploetic tip; *N* pneumatic cell in diploemmatic mastoid; *O* diploetic tip; *P* is part of occipital bone which takes part in formation of the jugular fossa. This specimen shows how a cellular invasion can take place into the tip by way of the retrofacial cells.

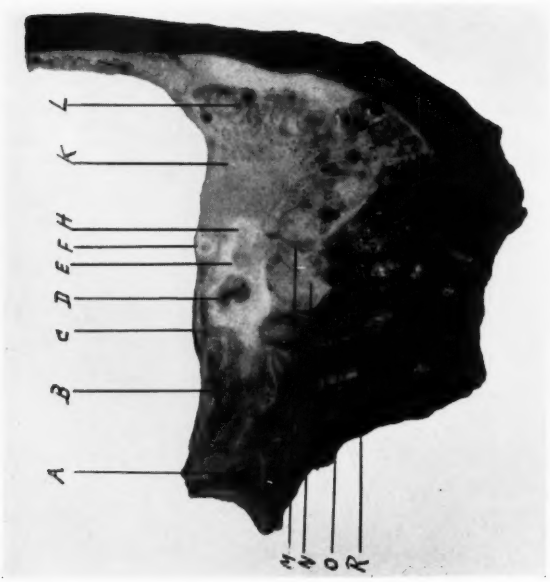

Fig. 17. Photograph of section through plane of facial canal. It is similar to Fig. 4 except in this specimen cells have been dissected posterior to facial canal as represented by the wire. It shows at *A* spheroid sinus; *B* diploetic tip; *C* cells communicating between tip and mastoid; *D* cerebellum; *E* vestibule; *F* superior semicircular canal; *H* external semicircular canal; *K* sclerotic part of mastoid; *L* pneumatic cells of mastoid; *M* facial nerve in canal; *N* bulb area diploetic in type; *O* internal carotid artery; *R* jugular vein. Notice cells communicating from mastoid posterior to facial canal to bulb area. Some cells have been dissected away as demonstrated by wire internal to facial canal. This specimen shows how extension from mastoid cells may occur by way of the retrofacial cells to bulb area and hence into carotid canal with subsequent invasion of the tip.

Empyema of the tip, when surgery is indicated, has extended to a location very close to the middle ear wall. The peritubal route is the natural course of evacuation of the empyema, rather than the plane of invasion. This opinion is based upon a careful study of this portion of the temporal bone (Figs. 10-13, 16). It is frequently demonstrated clinically by a secondary discharge of purulent material from a spontaneously formed fistula in patients with clinical evidence of purulent lesions in the apex. Lange is of the same opinion.[28] Streit,[32] Eagleton,[33] Myerson and his co-workers[34] have advised a subtemporal approach. Its value in comparison with the peritubal technic in empyematous lesions of the tip must be questioned, chiefly upon the basic principles of drainage. The writer is convinced that the subtemporal approach is definitely indicated in frank osteomyelitic pathology of the pyramid and apex (Figs. 20-22). Again we must return to the surgical pathology applicable to bony structures and apply our therapy to such pathology as may be present. Gimlet holes may suffice in localized purulent lesions, but osteomyelitis demands wide exenteration of the pathologic process, unless one is willing to assume the responsibility of future complications and sequelæ. The difference of opinion relative to the therapy of temporal bone lesions depends more upon the failure to recognize the concommitant pathology present than upon the technical method to be followed.

Gross observations such as presented may be subject to the criticism of the lack of microscopic correlation. It is the writer's belief that the most accurate observation, next to microscopic, is a study of the cellular character and anatomic location, by use of sections as described. It gives a comparative picture of the component parts, from which clinical and surgical conclusions may be obtained. A clear appreciation of the surgical anatomy of this structure not only frees the surgeon from anxiety during any technical procedure but enables him to forecast and follow the paths along which infection may spread through the bone (Fig. 23). The safety that comes of boldness and skill in surgical work cannot be attained without accurate anatomic knowledge.[35]

CONCLUSIONS.

1. From the observations as presented, the cellular character of a majority of temporal bones are of the mixed type.

2. This does not apply to the petrosal tip, which is predominantly diploetic.

3. Of the mixed type, the diplopneumatic is the most prevalent.

Fig. 20. Photograph of a dissection to demonstrate the approach to the petrosal pyramid and apex via the mastoid route. The anterior and posterior buttress have been removed. The specimen shows the mastoid tip, the facial nerve, the great superficial petrosal nerve, the middle ear, the cochlear portion of the petrosal pyramid, the middle meningeal artery with its anterior and posterior branch, the dura. By following the above mentioned surgical anatomy it can be readily seen that the dura may be lifted from the pyramid in the middle fossa so as to treat both the pyramid and tip by the indicated surgical procedure. By following the mastoid posteriorly the posterior fossa is approached, and the posterior surface of the pyramid may be treated accordingly.

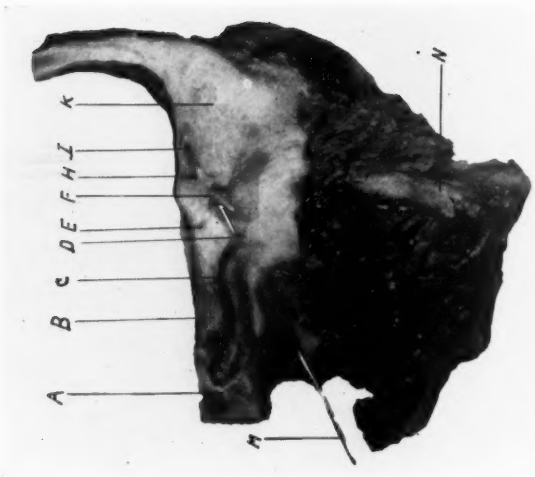

Fig. 19. Photograph of section through middle ear and carotid canal. It shows at *A* sphenoid base; *B* petrosal tip with Meckel's fossa; *C* carotid canal and vessel; *D* point between eustachian tube opening and carotid canal or point of entrance to peritubal cells to approach the tip; *E* part of cochlea; *F* middle ear with malleus and incus at *H*; *I* the mastoid antrum; *K* a sclerotic area of mastoid; *N* internal jugular vein; *M* probe in eustachian tube; it is in the tube above the cushion or bolster called the tubercles.

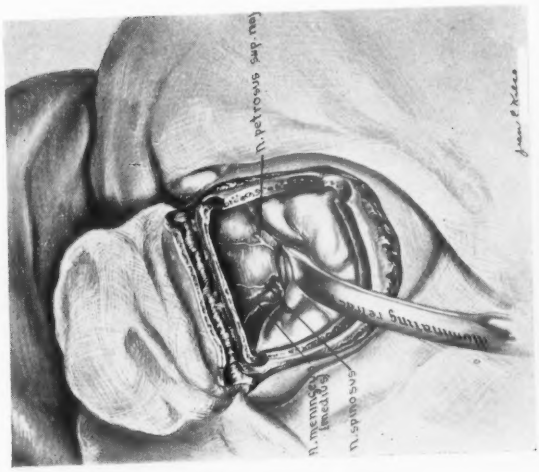

Fig. 22. Photograph to demonstrate method of approach to the pyramid and tip via a middle fossa approach. The dura is elevated with the elevator spatula. Though this demonstrates the osteoplastic approach, the muscle splitting of Cushing is to be preferred.

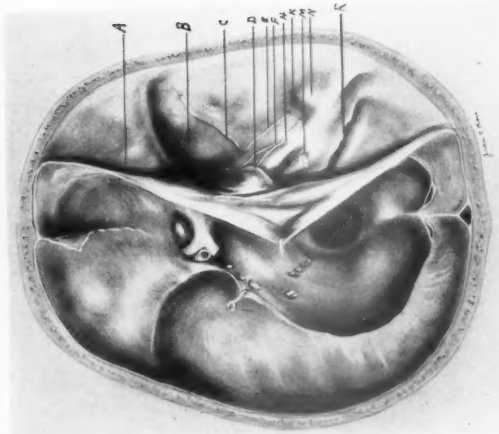

Fig. 21. Photograph to demonstrate surgical anatomy of the cranial fossa with dura retracted on right side. A mental picture of these structures is important in the subtemporal approach to the pyramid or tip, as well as by way of the posterior fossa. *A* the dura retracted; *B* the middle fossa; *C* the middle meningeal artery; *D* the gasserian ganglion with the foramen branches of distribution; *E* the external petrosal nerve entering the petrosal sphenoidal; *F* the small superficial petrosal; *G* the great superficial petrosal; *H* the subarcuate fossa; *I* the eminentia arcuata; *J* the auditory, facial and intermedius nerve; *K* the groove for the inferior petrosal sinus. The tentorium has been removed and the dura is retracted from the posterior fossa. The tip lies beneath and external to the gasserian ganglion.

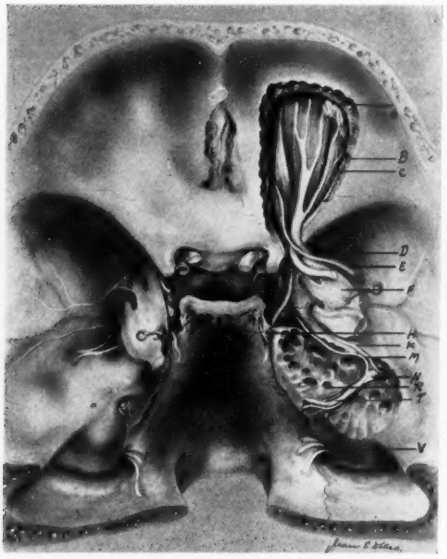

Fig. 23. A photograph of the surgical anatomy of the structures closely allied with the pathology and treatment of petrosal pyramid and tip lesions. *A* the lachrymal gland; *B* the orbit uncapped showing the muscles and nerves to same; *C* the abducens fibers to the external rectus; *D* the ophthalmic branch of the gasserian ganglion; *E* the fourth nerve; *F* the gasserian ganglion with the motor branch on the under surface in close association with the inferior maxillary division; *H* the abducens nerve passing through Dorello's canal beneath the petrosphenoid ligament; *K* the small superficial petrosal nerve; *M* the great superficial petrosal nerve; *N* the geniculate ganglion; *R* the petrosal pyramid with cellular character; *T* the facial, auditory and intermedius nerve passing through pyramid enclosed for a great distance in the dural covering; *V* the 9th, 10th and 11th cranial nerves entering the jugular fossa.

4. The cellular character of the mastoid is not a true index to other portions of the temporal bone.

5. The surgical anatomy as presented leads to the inference that the most prevalent route of invasion to the petrosal tip is from the mastoid by way of the posterior aspect of the pyramid, then by way of the anterior surface from the zygoma, or the peritubal or carotid canal directly from the middle ear, and finally by way of the retrofacial area via the bulb cells.

6. Petrosal tip pathology may occur by way of continuity of tissue or by vascular extension from infections in the middle ear or mastoid, also from pathology extraneous to the temporal bone.

7. The peritubal cells serve as a route of evacuation rather than one of invasion to the tip.

8. The logical approach for drainage to localized purulent pathology in the tip is by way of the peritubal cells, and for osteomyelitis, a subdural approach either from the mastoid or directly by way of the middle fossa.

9. The variance in the surgical anatomy of the temporal bone impresses one with the fact that clinical evidence, based upon the pathology present should be the guide to the type of therapy used.

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II.

THE EFFECT OF PHENOL IN HYPERESTHETIC RHINITIS WITH TISSUE STUDY OF THE NASAL MUCOSA.*

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The term "hyperesthetic rhinitis" is used to designate a condition characterized by swelling and pallor of the nasal mucosa accompanied by spasmodic sneezing, hypersecretion, obstruction to nasal breathing, itching or burning sensation in the eyes, nose and nasopharynx.

The etiologic factors are numerous. Attacks of hyperesthetic rhinitis may depend upon pollination of certain plants and trees, in which case they are seasonal, or they may have no relation to a pollination period and occur throughout the year. The first type is commonly known as hay fever, while the latter is variously called vasomotor rhinitis, allergic rhinitis, spasmodic rhinorrhea, perennial hay fever, etc. Sensitiveness to dust, feathers, animal or bird emanations, orris root, and certain food proteins are the common factors in this second type.

The persistence of symptoms in spite of active treatment and the frequent recurrence of attacks are outstanding features of hyperesthetic rhinitis to the extent that it is hardly possible to secure lasting relief. The great variety of treatment employed in the past suggests a lack of any particular method which is generally satisfactory except in the case of true hay fever or the seasonal type of hyperesthetic rhinitis. Because of the unquestioned success obtained by the allergists during the last twenty-five years in the treatment of hay fever and hay asthma, extensive research has been carried out in an effort to apply the same principles to the treatment of cases of hyperesthetic rhinitis not related to pollination. The etiology of many obscure conditions was clarified but unfortunately no specific therapy dependent upon these etiologic discoveries proved satisfactory in its application.

Besides the "allergic method," which included efforts to desensitize the patient and to eliminate the irritant, together with dietetic,

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

hygienic and climatic therapeutic measures, the following classified methods are of interest:

1. Physical measures, including the use of cautery, faradic current, galvanism applied to various sites of the body, diathermy coagulation, and ionization.

2. Internal therapeutic measures, which constitute a group of themselves and at the same time have been important adjuvants to other means of treatment. Here should be mentioned the administration of ephedrin, adrenalin, glandular extracts, calcium, atropin and its derivatives.

3. Local measures—the use of vasoconstrictors, the local use of astringents, caustics, irrigations and tampons saturated with solutions of choice.

4. Mechanical means, largely obsolete, such as the use of filters, nasal plugs and similar devices.

For the most part the above mentioned measures proved unsatisfactory. However, favorable results by the Warwick[1] method of ionization have been reported recently.

Our study concerns the use of phenol applied locally to the nasal mucosa in cases of hyperesthetic rhinitis. Phenol or carbolic acid is a weak acid possessing corrosive and poisonous properties. It gives a violet color with ferric chloride. It is a colorless, crystalline substance which becomes liquid on addition of 15 per cent water. It has powerful antiseptic and germicidal properties. It causes blanching of the skin and anesthesia. The blanching may be removed by the use of alcohol. The action on the mucous membrane is caustic and is followed by a localized necrosis varying in degree with the amount of the acid and the duration of its action.

Chemical cauterization in hay fever was employed as early as 1837 by Cazenave.[2] Thommen,[3] in his excellent history of hay fever, mentions the use of various chemicals such as carbolic, nitric and glacial acetic acids, by Brown, in 1884, and later by Logan, Clark, Taylor, Cheatham, Crippen and Douglas. Still later, in 1927, Killian[4] advocated the application of trichloracetic acid to "four sensitive spots" in the nose. More recently Chandler[5] reported the results obtained from the use of phenol in cases of hay fever. He reported excellent results for a period of one to two years and considered the treatment a preventive of common colds. He believed the carbolic acid desensitized the membranes. There was no disturbance of olfac-

tion nor evidence of poisoning. In 1933 Spivake[5] made an experimental study of sixteen cases of vasomotor rhinitis and two of asthma. Some of these cases were complicated by hay fever, others by chronic sinusitis with polypoid degeneration. He believed that the treatment blocked the nerve endings in the nasal mucosa, stopping afferent impulses from being carried to the nerve centers.

The use of phenol has much to commend itself to the competent rhinologist. The technic of application is simple, saving time and expense. The patient is not subject to a long series of sensitization tests, the result of which is often inconclusive. He is also saved a long series of inoculations with sera and specific desensitizing extracts, in case some specific factor is found. On the part of the well trained rhinologist the treatment has appeal in that it does not require elaborate and expensive apparatus, such as is used in ionization and diathermy.

With these facts in mind, the effect of phenol was studied in a series of thirty consecutive cases, suffering from hyperesthetic rhinitis. The study included detailed analysis of the history, careful diagnosis, close clinical observation of the patient, pathologic examination of the nasal mucous membrane before and after treatment and a record of the changes in the volume of air exhaled, as registered on a breathing plate.

The technic of application was as follows: A thorough application of concentrated phenol, U. S. P., was made to the entire nasal mucosa, excepting the olfactory area. In some cases an application of 2 to 5 per cent cocain solution was made to the membranes prior to the treatment. Vaselin and alcohol were used to protect the vestibule and the mucocutaneous margins of the nose. Care was exercised to avoid the nasopharynx lest some of the solution affect the pharynx and larynx. In six cases a second application was made.

Nearly all the cases had previously submitted to various types of treatment without relief, with the exception of certain hay fever cases who had been helped to a degree by pollen extracts. During the course of our study all therapeutic measures, other than the application of phenol as described, were omitted with the exception of one patient who received two pollen extract injections just prior to the phenol treatment.

The immediate effect on the nasal mucosa was coagulation, with an appearance of grayish, white discoloration, followed by an acute edema and obstruction with hypersecretion. The patient experienced a sense of stuffiness, heaviness in the head, or headache and sneezing

attacks. The sneezing and hypersecretion began to diminish in one to two hours and the clogging and congestion usually continued from twenty-four to forty-eight hours. The appearance of the membrane during the period of reaction showed congestion, swelling and edema, watery or inspissated discharge with the presence of shreds and plaques of sloughing mucosa and at times abrasions of the membrane.

The following case summaries are characteristic of subsequent events:

REPORT OF CASES.

CASE 1.—R. C., male, aged eleven years. The patient suffered from hyperesthetic rhinitis for eight years, asthma for five years and fall hay fever for one and a half years. He had allergic treatment for asthma and hay fever for a year and a half without results. One phenol application was made on February 13, 1934. This kept him free of symptoms until August 16, when the usual hay fever attack started. Phenol was reapplied on August 30, 1934. Ragweed injections were discontinued and the patient had a comfortable hay fever season.

CASE 2.—E. H., female, aged forty-four years. The patient suffered from hyperesthetic rhinitis since childhood. The complete nasal obstruction was very distressing but sneezing and discharge were not so marked. She has been found sensitive to dust, feathers, tobacco, goat hair and several foods. She also suffered from chronic maxillary sinusitis. The patient had allergic and local treatment and an operation fifteen years ago without results. Phenol was applied on May 5, 1934. Burning of the eyes and headaches followed for two days. On the third day this reaction disappeared. She now has an open nose for the first time that she can remember. To date she feels fine, has no clogging, no sneezing and a very slight discharge, probably due to the chronic sinusitis.

CASE 3.—L. D., female, aged twenty-eight years. The patient has suffered from hyperesthetic rhinitis since last winter. No skin tests have been done, nor is there any allergic history. Phenol was applied on July 5, 1934. She had two days of discomfort followed by cessation of nasal blockage and hypersecretion. There has been no recurrence of symptoms in two months and the nose shows good breathing passages.

CASE 4.—R. C., female, aged forty-four years. This patient has suffered from hyperesthetic rhinitis and spring hay fever for over ten years. She has obstinate headaches. Skin tests revealed sensitivity to wool and dust. Her father and brother have asthma and her daughter has urticaria. Phenol was applied on April 5, 1934. A few days later the patient had complete relief from her symptoms, and on June 5, 1934, she asked for a reapplication of phenol to be sure that the hay fever season would not bring back her trouble. She was seen again on September 8, 1934, and reported that she had been free from any symptoms of hyperesthetic rhinitis or hay fever but that her headaches still troubled her at times. The very thick middle turbinates were fractured away from the septum and crushed to relieve pressure.

CASE 5.—H. W., male, aged thirty-seven years. The patient has suffered from seasonal hay fever for three successive years. He was found to be sensitive to rag-

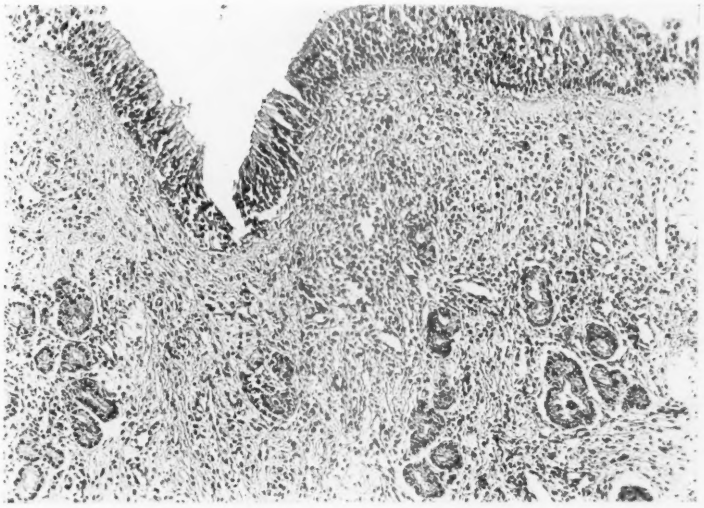

Fig. 1. Before phenol treatment. Showing edema of submucosa, with glands situated farther from the epithelial surface than in the normal. The glandular acini also are widely separated.

weed and dust. His mother has the same trouble. Patient came to the clinic in mid-season complaining of severe attacks of sneezing, clogging of the nose and profuse watery discharge. Phenol was applied on August 30, 1934. Slight discomfort followed for two days and then symptoms disappeared. "No trace of hay fever two weeks after application." The nasal membranes were pink, there was no hypersecretion and the breathing space was good. The patient showed no symptoms of hay fever throughout the rest of the season.

A study of the cases as a group showed the following: The age range was from five to fifty years. There were thirteen males and seventeen females. Some hereditary factor was present in six cases.

In respect to type, eight cases were of the seasonal variety, fourteen were perennial and eight were combined type. Three of these were suffering from asthmatic symptoms. Twelve patients had inflammation of one or more sinuses, nine had deviated septa, and one, nasal polyps.

The volume of expired air was measured on a breathing plate just before treatment and at intervals following treatment in a group of eleven cases. When first seen certain patients had complete blocking of both nares, others complete blocking of one side and partial blocking of the other.

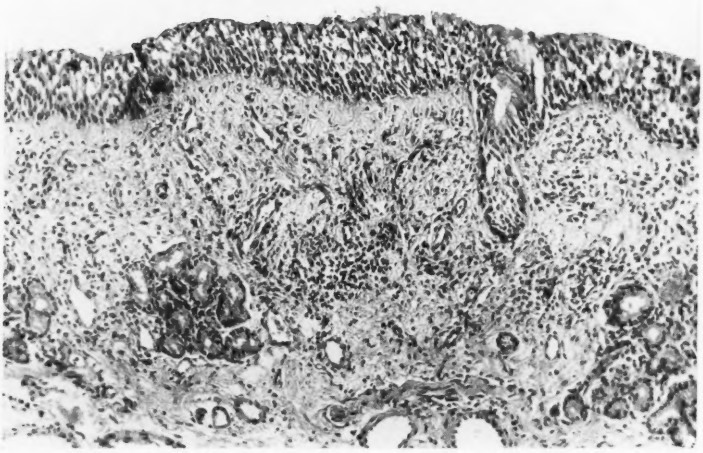

Fig. 2. After phenol treatment. Less edema with the glands situated closer to the overlying epithelium. More fibrous connective tissue present in the submucosa.

Of the thirty cases studied, twenty-four, or 80 per cent, showed definite improvement and twelve were free from symptoms for periods of one to nine months. Two cases were unimproved and four doubtful, because of incomplete follow up. Observations upon this group of cases and upon others are being continued.

Thirty-one sections of the nasal mucosa were removed from the inferior turbinates for a microscopic study. The summarized pathologic report follows:

"An accurate comparison of the histologic findings in the sections of the nasal mucous membrane removed from the inferior turbinates prior to and following the application of phenol is rather difficult. Although the tissues were carefully removed they varied considerably, especially the thickness of the epithelium and the number of glands present in the submucosa. The patients from whom the mucous membrane was removed were of different ages and sex and all presented a somewhat different picture, especially the amount of mucus in the epithelium and glands, the amount of edema and extent of the inflammatory exudate into the submucosa. The time interval of removing the tissue following the application of phenol varied as much as from five to thirty-three days in different individuals.

The following significant differences, however, were noted:

1. The majority of the sections removed before the treatment stain quite normally with hematoxylin and eosin, whereas in those taken after phenol the nuclei stain rather intensely with hematoxylin while the acidophilic elements take the eosin stain very poorly.

2. The pseudo-stratified ciliated epithelium of the former group forms a rather flat compact tissue through which are scattered well stained goblet cells, the free or cilated edge being quite well preserved. The corresponding surface epithelial cells of the latter group are more widely separated, contain many vacuoles and the mucus stains much less intensely.

3. The sections removed following treatment with phenol show less edema, the inflammatory exudate is somewhat more extensive and there is definitely more fibrous connective tissue proliferation in this group.

4. In the section taken after treatment the glands in the submucosa are less numerous, contain less mucus, and stain less intensely with eosin than the epithelium. The individual acini are somewhat more widely separated and less uniform in size; some are considerably dilated and show evidence of degeneration of epithelium.

5. The most striking differences are to be seen in the vascular spaces. In general, the capillaries and lymphatics in the first group are more numerous and more open. The small vessels and the larger sinuses, in the biopsies removed prior to the treatment, contain much more blood than those in the biopsies removed after treatment, in which there is generally less blood. The sinuses are quite collapsed and the endothelial lining is degenerated in many instances."

SUMMARY.

The effects of intranasal application of phenol (carbolic acid) in hyperesthetic rhinitis were studied in thirty cases. Microscopic examination of thirty sections of tissue removed from the nasal mucosa was made. The volume of air expired after treatment was compared to that expired previous to treatment by plotting of graphs from a breathing plate.

CONCLUSIONS.

Local application of phenol to the nasal mucosa in cases of hyperesthetic rhinitis is of value in both the seasonal and perennial types.

Improvement is manifest by partial or complete relief from attacks of sneezing, watery secretion and blocking of the nares.

Microscopic study of the nasal mucosa following the application of phenol shows an increase in the connective tissue (fibrosis) together with a diminution of edema and of vascularity.

Acknowledgment is gratefully made to Dr. F. Vistreich and Dr. D. M. Angivine for their valuable assistance.

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III.

LYMPHATIC DRAINAGE OF HEAD AND NECK— EMPHASIZING SPECIAL STRUCTURES.*

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All the structures of the head and neck have their lymphatic drainage either directly or indirectly into the superficial and deep cervical lymph glands. The superficial cervical glands are arranged along the course of the external jugular vein and its tributaries and around the superficial structures of the anterior triangle of the neck. The efferent vessels of these glands drain the lymph into the deep cervical glands. Since we are concerned chiefly in this discussion with the lymphatic drainage of the deeper structures of the head and neck, a detailed description of the superficial glands will not be necessary. However, special groups of superficial glands will be described where they are concerned with the drainage of certain structures. The deep cervical glands are arranged along the carotid vessels and are divided into superior and inferior groups.

The superior group includes all those glands related to the carotid arteries which lie above the omohyoid muscle. We may recognize a medial group, situated in front of the carotid vessels, and a lateral group, situated behind those vessels. The upper medial group receives afferent vessels from the nose, paranasal sinuses, face, mouth, tongue, ear, tonsil, upper parts of the pharynx and larynx, upper part of the thyroid gland, submaxillary, sublingual and parotid salivary glands, and from the interior of the cranium. The efferents from these glands pass both to the inferior deep cervical glands and to the jugular trunk. The upper lateral group receives lymph from the ear, the posterior part of the scalp, the superficial cervical glands, the brain, and the suboccipital region. These glands are intimately connected with the medial group, and their efferents pass to the inferior deep cervical glands and to the jugular trunk (Fig. 1).

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*Presented before the meeting of the Mid-Western Section of the American Laryngological, Rhinological and Otological Society at Dallas, Texas, January 28, 1935.

Department of Anatomy, Baylor University College of Medicine, Dallas, Texas.

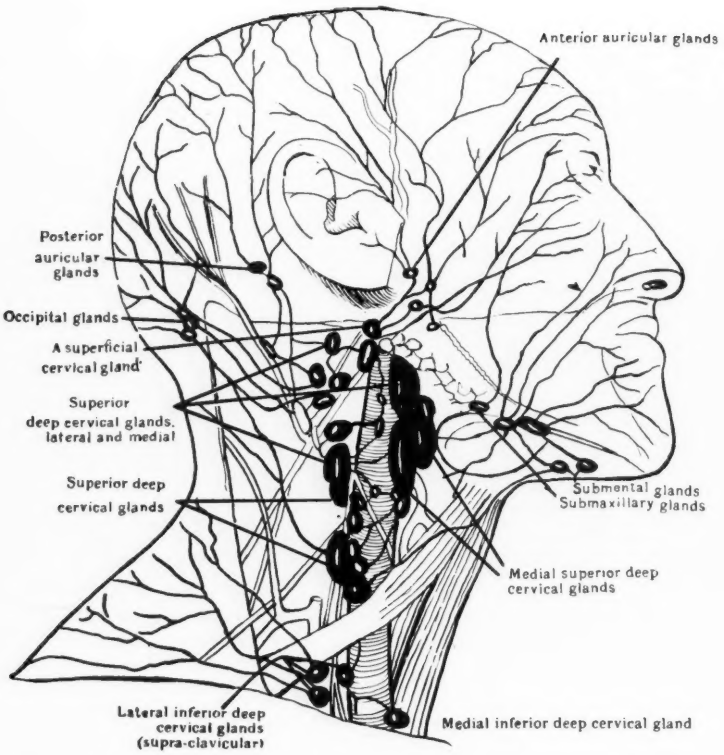

Fig. 1. Lateral view of the head and neck showing the general arrangement of the lymphatic glands and ducts.

The inferior deep cervical lymph glands (supraclavicular) lie below the omohyoid muscle and are divided into a medial and a lateral group. The medial group is closely related to the terminal portion of the internal jugular vein. These glands receive afferents from the upper deep groups, from the pretracheal and paratracheal glands, and from the upper part of the thorax. The lateral inferior cervical glands lie in the subclavian triangle, over the third part of the subclavian artery. They receive afferents from the lower parts of the neck, larynx, pharynx, thyroid gland, upper part of the trachea, the upper part of the thorax, and from the deep portion of the mammary gland.

The lymph vessels from the external ear and meatus pass to the anterior and posterior auricular glands, which are situated immediately anterior and posterior to the auricle, respectively, and these

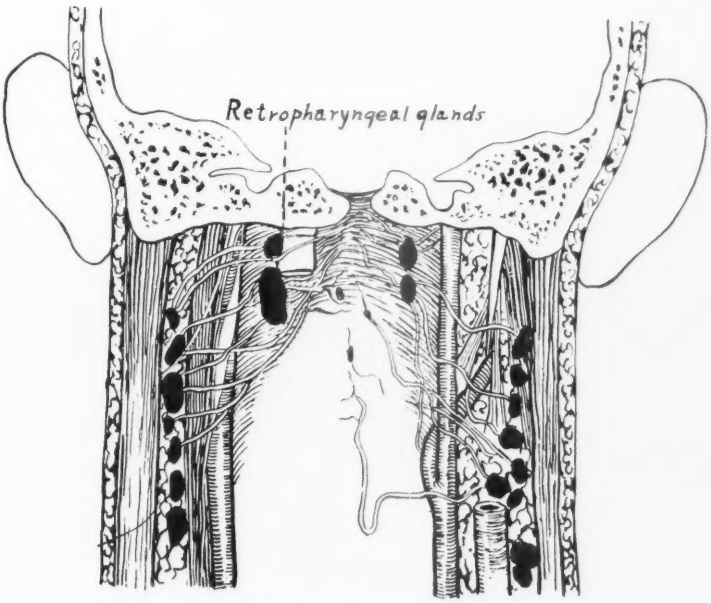

Fig. 2. Diagram of the retropharyngeal lymph glands.

glands send efferents to the superficial and superior deep cervical glands. The efferents from the lateral parts of the middle ear pass to the posterior auricular glands, and from the medial part of the middle ear and auditory tube they pass to the retropharyngeal glands. The retropharyngeal group consists of a number of glands situated in the loose connective tissue lying between the posterior wall of the pharynx and the superior portion of the prevertebral fascia (Fig. 2). Both of these groups send efferents to the superior deep cervical glands.

The lymph vessels of the external nose form two groups, namely, superior and inferior. The upper group of ducts ends in the anterior auricular glands, and from here efferents pass to the parotid and upper deep cervical glands. The inferior group of vessels from the nose passes to the submaxillary lymph glands, and these glands in turn send efferents to the superior deep cervical glands. The lymph vessels of the upper and lower parts of the nose communicate with one another to a certain degree but not freely. The lymph vessels from the anterior part of the interior of the nose pass to the submaxillary glands, and those from the posterior part go to the retropharyngeal and medial superior deep cervical glands. The submaxillary glands are found in the submaxillary triangle immediately below

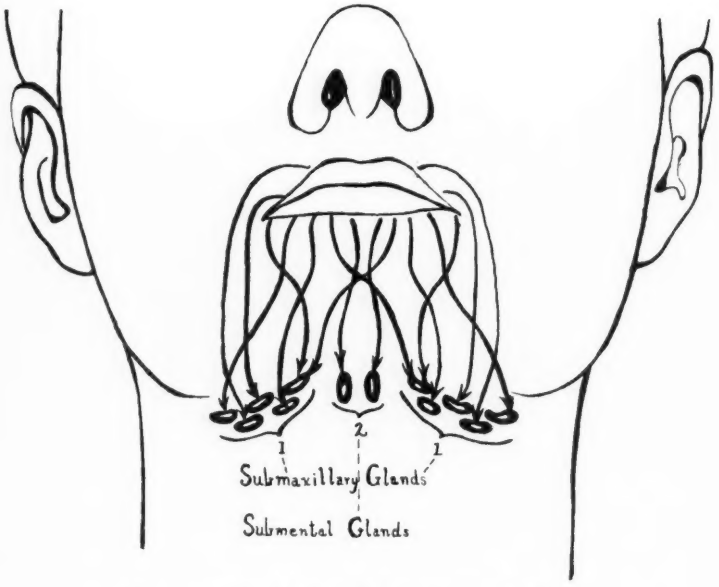

Fig. 3. Diagram of the lymphatic drainage of the lips.

the body of the mandible and in close relation to the submaxillary salivary gland. The retropharyngeal and submaxillary glands drain into the superior deep cervical glands. The lymph drainage of the paranasal sinuses is not definitely known, but it has been suggested that vessels from these structures pass, like those from the nose, into the superior deep cervical and retropharyngeal glands.

The lymph vessels from the medial part of the lower lip pass to the submental glands and occasionally directly to the superior deep cervical glands. The submental group consists of two or three small glands lying in the superficial structures directly below the symphysis of the mandible (Fig. 3). Efferents from the submental glands terminate in the upper deep cervical glands. Efferents from the remaining parts of the lower lip unite with those from the upper lip and pass to the submaxillary lymph glands. From here lymph goes to the upper deep cervical glands. Some of the lip vessels may pass into the superficial cervical glands.

The lymph vessels from the cheek terminate in the submaxillary and superficial and superior deep cervical lymph glands.

Lymph vessels from the outer part of the anterior portion of the mandibular gum end in the submental glands. Those from the pos-

terior part, together with those from the outer part of the maxillary gum, pass to the submaxillary glands. The vessels from the inner part of the gum of the mandible end in the submaxillary glands, while those from the inner part of the gum of the maxilla and the hard and soft palates end in the medial superior deep cervical glands.

It is probable that the lymph vessels from the teeth of the mandible, and mandible itself end in the submaxillary glands, and those from the teeth of the maxilla pass to the infraorbital glands, although this is not definitely known. Efferents from the latter group of glands pass to the anterior auricular and submaxillary glands.

The lymph vessels of the tongue are arranged in four groups—anterior, middle, posterior and deep. The anterior and middle groups communicate with each other and with those of the opposite side. The anterior group drains the tip of the tongue and the lower free portion and passes to the submental glands. The middle group drains the anterior two-thirds of the tongue, except the tip, and terminates in the submaxillary and superior deep cervical groups. The posterior vessels of the tongue drain that part of the tongue posterior to the vallate papillæ and end in the superior deep cervical group. Vessels from the deep central part of the tongue go to the superior deep cervical glands (Figs. 4, 5).

The lymph vessels of the parotid gland end in the parotid and upper deep cervical glands. Vessels from the submaxillary salivary gland terminate in the superior deep cervical glands and not in the submaxillary glands as might be expected. It has not been definitely ascertained to what groups of glands the lymphatics of the sublingual glands pass; but it has been assumed that many of them join the vessels from the tip and free portion of the tongue, while others pass directly to the submaxillary glands.

The vessels from the upper and lateral parts of the pharynx drain into the retropharyngeal glands. Those from the anterior and lower parts of the pharynx pass to the upper medial group of cervical glands.

Lymph vessels from the palatine tonsil and the adjacent parts of the pharynx pass to the medial upper deep cervical glands. One gland, termed the tonsillar gland, which lies over the common facial vein in the digastric triangle, receives lymphatics directly from the palatine tonsil. Its efferents end in the medial group of superior deep cervical glands. This is the first gland that can be palpated in cases of acute tonsillitis.

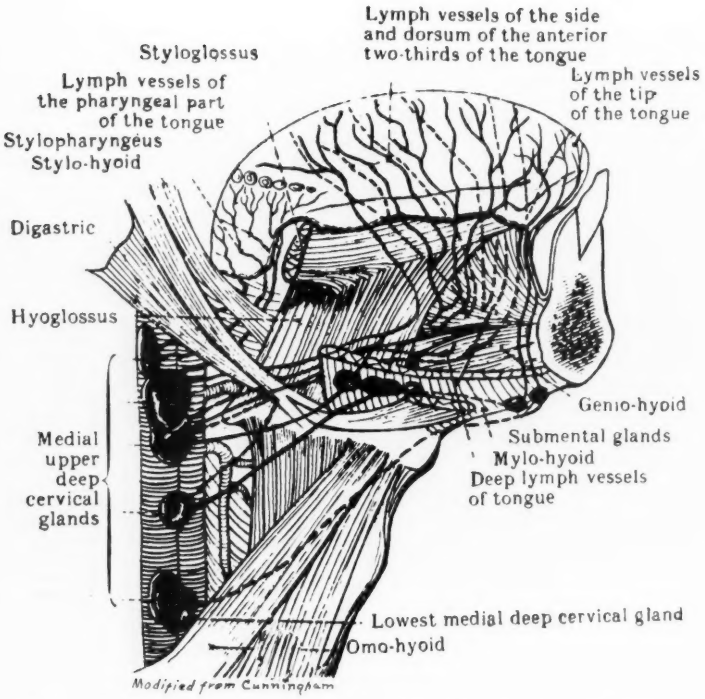

Fig. 4. Diagram of a lateral view of the tongue showing the arrangement of the lymphatic ducts.

The lymph vessels which drain those portions of the larynx above the vocal folds pass to the upper deep cervical glands. The vessels from the anterior part of the lower portion of the larynx pass through the cricothyroid ligament and end in the laryngeal, pretracheal and upper deep cervical glands, and these glands drain chiefly into the lateral group of inferior deep cervical glands. The efferents from the posterior part of the larynx terminate in the retropharyngeal glands. The lymph vessels of the upper and lower parts of the larynx communicate very sparingly over the vocal cords, but fairly freely in the posterior wall of the pharynx. This arrangement of the lymphatics of the larynx accounts for the edematous condition frequently encountered in the upper part of the larynx while the lower part of that structure remains uninvolved.

Although, strictly speaking, only the superior portion of the trachea is a neck structure, yet the lymphatics of the entire trachea

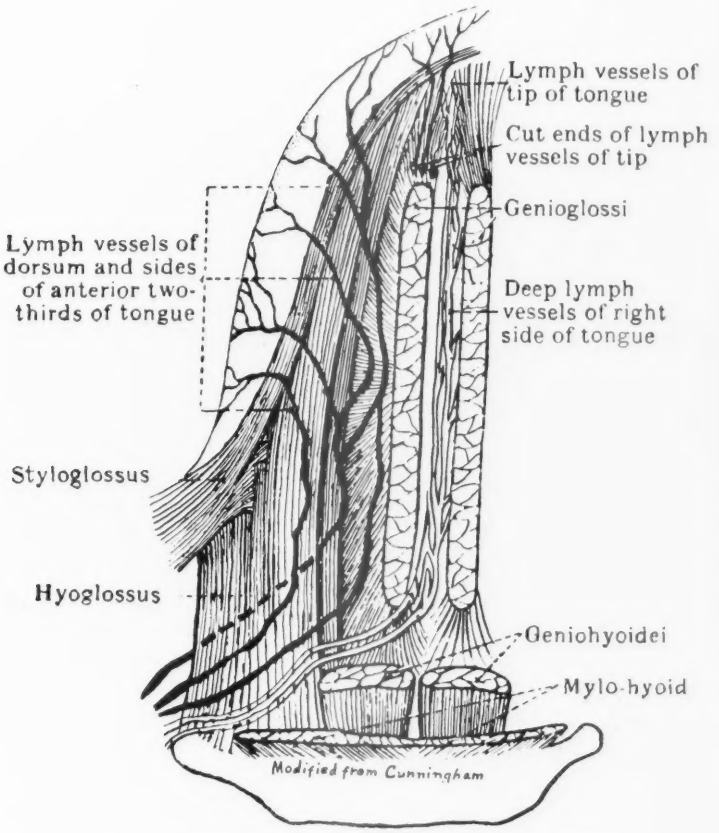
Lymph vessels of tip of tongue
Cut ends of lymph vessels of tip
Genioglossi
Lymph vessels of dorsum and sides of anterior two-thirds of tongue
Deep lymph vessels of right side of tongue
Styloglossus
Hyoglossus
Geniohyoidei
Mylo-hyoid
Modified from Cunningham

Fig. 5. Diagram showing the deep lymphatics of the tongue.

and its subdivisions, the bronchi, are so intimately connected with one another that it does not seem out of order to give at this point a brief description of the lymphatics of all these structures. This discussion is based chiefly on the description given in Cunningham's Textbook of Anatomy, sixth edition.

The pulmonary lymphatic glands are found in the substance of the lung, between the bifurcations of the smaller divisions of the bronchi. These glands receive afferents from the lung substance and send efferents to the bronchopulmonary glands.

There are two groups of bronchopulmonary glands, situated one each in the hilus of the lung and around the bifurcations of the

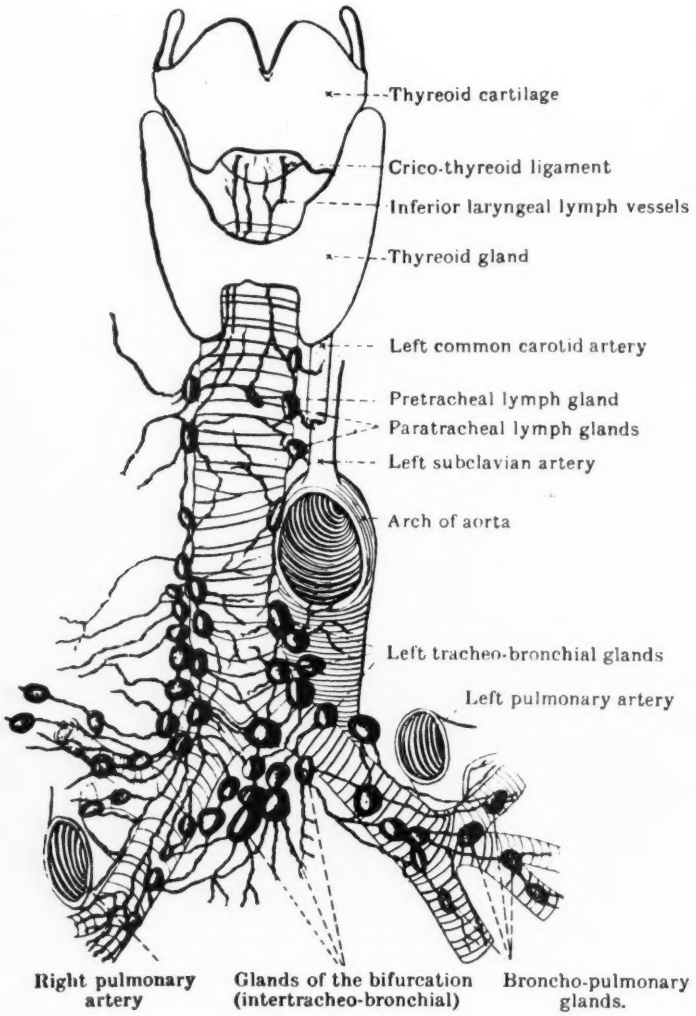

Fig. 6. Diagram of the lymph glands of the trachea and bronchi (modified from Cunningham).

bronchi. They receive lymph from the lung substance, the pleura and from the bronchopulmonary glands. Their efferents pass to the tracheobronchial and intertracheobronchial glands (Fig. 6).

The intertracheobronchial glands lie below the trachea and in the angle formed by the trachea and the two bronchi. These glands receive vessels from the bronchopulmonary glands of both sides, from the heart and adjacent structures, and from the posterior mediastinal lymph glands. The efferent vessels of these glands terminate in the tracheobronchial glands of each side.

The tracheobronchial glands lie in the angle between the trachea and bronchus of each side. Lymph is received by these glands from the other bronchial glands, from the trachea and bronchi, and from the heart and mediastinum. The efferent vessels of the left group of glands enter the thoracic duct and communicate with the left medial inferior deep cervical glands. The right group of glands drains chiefly into the right bronchomediastinal trunk, and this duct joins either the right innominate vein or the right lymphatic duct. The bronchomediastinal glands of the two sides are intimately connected with one another and with the paratracheal glands.

The paratracheal glands lie on either side of the trachea and are connected over the surface of the trachea with the pretracheal glands. The paratracheal and pretracheal glands drain the upper part of the trachea, a portion of the thyroid gland and the lower compartment of the larynx. Their efferents pass to the tracheobronchial glands and to the inferior deep cervical glands.

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IV.

THE PROBLEM OF THE ETHMOID.*

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Before taking up the details of my subject I should like to say a word as to the reasons for its selection. I think it was in 1920 that I came to Houston with my friend, the late Dr. Ross Hall Skillern, who had been invited to present a paper before the Eye, Ear and Throat Section of the Texas State Medical Society, under the chairmanship of Dr. John H. Foster, and the chairman very courteously asked me to take part in the discussion. Dr. Skillern's subject was similar to the one I have chosen, as was also his address before the British Medical Association in 1928, which I also had the privilege of discussing. Much water has gone over the dam since 1920, so I thought that I could do no better at this time than attempt to bring certain phases of this subject up to date.

Eugene R. Lewis well says that "There is no reason for believing that certain therapeutic indications or agencies apply particularly to ethmoiditis but not to troubles of similar nature in, say, the frontal, the antrum or the sphenoid; in other words, terms such as 'ethmoiditis' and 'sphenoiditis' denote only anatomic differences in site of manifestation; it is, therefore, not believable that treatment of one kind is indicated in sinusitis manifested in ethmoid areas, whereas treatment of another kind is indicated in troubles of similar nature manifested in other areas."

Now while this statement is true when viewing sinus disease as a whole, and when considering general theories of etiology, pathology, prophylaxis and treatment, nevertheless anatomic differences of structure do play a large part in different areas, instances of isolated infection being by no means uncommon, and for purposes of discussion it is certainly more convenient to divide diseases of the accessory nasal sinuses into different groups, more especially when considering surgical measures. Whatever may be the practice in the future, the time has not yet arrived when surgery can be ignored in

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the treatment of sinus disease, although it must never be forgotten that the physiologic function of the nose must be preserved as far as possible. This is a sound underlying principle, and restoration of, not damage to, this function should be the ideal, even if not always obtainable, in any line of treatment advocated.

This essay must deal, almost exclusively, with chronic ethmoid infection. Acute ethmoiditis is a very common thing—as common indeed as the prevalent "cold," of which it is a sequel if not an actual part. Few, if any, "colds"—i. e., infection of nasal mucous membrane, run a course of more than a few hours without ethmoid and other sinus involvement. We are so used to the symptomatology of an acute rhinitis or coryza that little attention is paid to its usual extensions. Cure of the rhinitis leads to cure of the sinus involvement in most instances. Abnormalities of nasal development, septal deviations in the ethmoid region, previous ethmoid disease with turbinal hypertrophies or hyperplasias, inadequate or obstructed cell ostia, prolong the sinus involvement and make for chronicity, if this is not already established. Previous or chronic ethmoid involvement is an important etiologic factor in the production of repeated attacks of acute infections which frequently add to the pathology of the chronic condition. Systemic diseases, allergies, vitamin deficiencies, poor health habits, poor personal hygiene, all play important parts in the production of repeated attacks of acute infection and in the continuation and increasing of chronic ones. Treatment of the acute condition should obviously be of the most conservative nature that the situation allows, and consists in measures calculated to obtain shrinkage of swollen membranes with resultant re-establishment of ventilation and drainage, the physiologic function without which cure cannot be established. Between attacks, attention to systemic causative factors should be instituted and obstructive lesions in the nose corrected or eliminated, proper breathing and bodily exercises instituted and the general welfare of the body attended to. Surgery is rarely indicated in a purely acute attack, unless the disease has extended into other structures, such as the orbit, and grave damage threatens. Even then it should be of the least destructive type that will relieve the symptoms. When the infection is confined to the ethmoid capsule and is entirely of the acute variety, the only surgical procedure permissible, and one which is entirely nondestructive, is fracture of the middle turbinate towards the septum in its anterior half when obstructing ventilation and drainage of the anterior series of cells, or breaking it towards the lateral nasal wall in the posterior half to promote these factors in the spheno-ethmoid recess. Tur-

binectomy is not indicated in acute infections confined to the ethmoid cells and is, moreover, a mutilating and dangerous procedure. Physical therapy, particularly heat, preferably administrated with the infra-red lamp, often gives marked relief, presumably by stimulating peripheral circulation and restoring defective ciliary action.

The old division of chronic ethmoiditis, or sinusitis in general, into hyperplastic (or catarrhal) and suppurative, is convenient for purposes of description and classification, but is often inaccurate, since many sinus infections, mainly characterized by hyperplastic mucous membrane and polyp formation, are complicated by suppurative discharges. An ethmoiditis that is first recognized as an hyperplasia, which is an infectious process, may readily prove a fertile incubator for secondary infections because of decreased or destroyed ciliary and glandular activities, and by obstructed ostia, the case thus becoming the mixed variety.

Since Skillern's presentation, a change has taken place in our conception of the relationship existing between sinus disease and allergic states. Many years ago, before the study of allergy was a definite specialty and before the name had even been coined, it was definitely understood, although the mechanism was unknown, as indeed it still is, that surgery of the middle turbinates, the removal of polypoid masses, and certain sinus operations, would at times relieve patients suffering from hay fever and asthma. In recent years it has been reported by competent and reliable surgeons that removal of hyperplastic sinus linings, notably from the maxillary and ethmoid cells, relieved patients from asthmatic attacks, sometimes for years, or until there was a recurrence of the sinus infection. It has been shown definitely that a study of the cytology of the nasal secretion shows large numbers of eosinophiles to be present, and Ersner has demonstrated equally large numbers in the mucosa and submucosa of the lining membrane of the maxillary sinus in asthmatics. It is reasonable to suppose that the same condition exists in the other sinuses as well.

When the use of radiopaque substances was first introduced for the roentgen ray study of the thickness of the lining membrane of sinuses it was noted by Proetz that a maxillary sinus that at one observation was almost completely occluded by swollen mucosa might, a few hours later, show a membrane of only normal thickness. This condition could only be of allergic origin, and readily explained the fact observed from time to time by different surgeons, to their chagrin, that on opening an antrum, opaque to the x-rays a few days before, an apparently normal membrane was discovered. These observations have forced upon us rhinologists a further study of these

phenomena, which is still in progress, with the old question, as yet unanswered: "Does the sinus infection cause the asthma, urticaria, etc., or is the allergy responsible for the sinus condition?" To say that the systemic manifestations are due to a bacterial allergy of sinus origin has always seemed to me, perhaps wrongly, I not being an allergist, to be begging the question.

Recently Kern and Schenck have written extensively to show that allergy is a constant factor in the etiology of so-called mucous polyps springing from the ethmoid. They, at present, differentiate between (1) the mucous polyp, edematous fibroma or, as they prefer to call it, myxoid fibroma; (2) mixed polypoid hyperplasia and (3) papillary hypertrophy or mulberry polyp. It is those in the first group only that are considered to be of definitely allergic origin and evidence is presented to show that allergy, albeit a true hypersensitivity to bacteria, is the causative factor in all such polyps. In their experience, mucous polypi are extremely common in allergic conditions of the respiratory tract, and are rare in nonallergic conditions, even in extensive and chronic sinus infection. All polyp cases give personal histories of allergy, often overlooked and often a familial history as well, the polypi occur usually in the protracted and perennial types and much less frequently in the seasonal, and the presence of large numbers of eosinophiles in the secretions, the tissues or the blood denotes recent contact with the causative allergen. What remains to be proven, it seems to me, is whether or not, if we accept this theory of polyp causation, we can apply it to all sinus hyperplasias. I believe that this is quite probable and will give us another definite point of attack, although it will not render obsolete other accepted forms of treatment. Furthermore, it must not be overlooked that allergic sinus disease often is accompanied by true sinus suppuration, and is, indeed, frequently the cause of the latter, because of the resulting obstruction to ventilation and drainage.

The widespread use of radiopaque substances in the roentgenologic study of the sinuses, so ably brought forward by Proetz, has greatly added to the value of our diagnostic methods. The displacement method of introduction, advocated by Proetz, gives us much diagnostic information if properly carried out. Failure to enter a sinus suggests an occluded ostium, while failure of prompt evacuation denotes destroyed cilia or defective ciliary action. Study of the films not only shows us the size, shape and extensions of the cells being studied, but also the presence of intrasinus polypi and the thickness of the lining mucosa, and also, if serial films are taken at close intervals, whether the thickening of the mucosa remains constant or

varies greatly, the latter suggesting allergy. The method is so simple that anyone can avail himself of it, and all of the sinuses, including the frontals and maxillaries, can be sufficiently filled at one sitting for a very complete diagnosis, albeit many radiologists do not feel the need of this method for arriving at a roentgenologic diagnosis. Aside from disappearing allergic swellings, the roentgen ray pictures may be misleading in other respects. Formerly it was of common occurrence to receive reports of extensive ethmoid disease in the absence of diagnostic clinical signs. This is less common now, due to improvements in technic, but the overlying cells in this area usually make for confusion and difficult interpretation. The individual scarcely exists who has not had repeated attacks of ethmoid infection, which in time make permanent tissue changes which give signs of altered densities, which may mean increased mucosal thickness with consequent smaller aeration spaces, absorption or thickening of the bony walls. Frequently these changes in density are reported to us as being due to present disease, whereas, in the absence of clinical findings, they should be considered only as signs of acute attacks, maybe frequently repeated, of disease long past.

Underlying etiologic factors must always be sought for and eliminated or counteracted for the successful management of any case of sinusitis, or of ethmoiditis in particular. Lewis says that "purulent discharge from a sinus may be the expression of a general systemic, metabolic or nutritional wrong or of local tissue reaction to a foreign body, toxin or trauma, any of which may reduce local resistance sufficiently to give foothold to bacteria for otherwise impossible invasion of the tissues; with the removal of the chief etiology, the tendency is toward spontaneous restoration of the individual to normal. In a similar manner mucosal tissue reaction to allergens or vitamin deficiency may secondarily give focal footholds to indigenous infections."

What are such general conditions of bodily health that must be looked for? Allergy has already been commented upon. Avitaminosis, or vitamin deficiency, has been shown by the experiments and observations of Shurly and others to be a frequent factor. Individuals, particularly children (and experimental white rats), receiving insufficient supplies of vitamins A and D, easily succumb to bacterial nasal infections, acute "colds" and acute or chronic sinus disease. Metabolic faults should be investigated and anemias combatted. Defects in general and bodily hygiene must be sought for and corrected, as they are all potential contributing causes. Furthermore, the sinus infections of children, as Dean has shown, are often largely contributed to, if not caused by, tonsil and adenoid infections. This

takes place primarily because of the obstruction to nasal respiration that is usually present, with its accompanying congestion and engorgement of the normal mucosa with consequent obstruction to drainage and aeration, and also because of the location of a near by constant primary source of infection, which, under the imposed circumstances, may easily reach the sinuses by contiguity or continuity. On the other hand, it is quite possible that sinus disease, once established, will lead to frequent attacks of acute tonsillitis and adenoid infection, thus completing a vicious circle. The local defenses of the sinuses are great if given free play. They are, first, the normal action of the ciliated epithelium and the glandular secreted blanket of mucus, when combined with unobstructed facilities for drainage and ventilation, the phagocytic action of the white cells of the blood stream and the connective tissue phagocytes, the histiocytes, of the reticulo-endothelial system, as pointed out by Fenton.

Why does a case of chronic ethmoiditis come to the rhinologist —for what is he seeking help? He comes for one of two main reasons: 1. Persistent, or intermittent, nasal discharge, either anterior or posterior, possibly a bad breath that his friends complain of, frequent attacks of long continued "coryza," headache (although Shambaugh says this is uncommon), nasal obstruction, a feeling of fullness in the head, or aphrosexia. 2. He is sent in by the ophthalmologist, internist or pediatrician, with a request for a search for, and elimination of, if found, a focus of infection for a case of eye inflammation that does not yield to usual methods of treatment, arthritis, bronchiectasis, colitis, gall bladder infection or any toxemia or anemia, retardation of development, or, in the case of our own specialty, chronic laryngitis or tracheitis. In either case therapeusis and a quick cure is demanded and expected, in the first instance by the patient, and in the second place by the referring physician, without due understanding of the pathology and conditions to be counteracted. In either case the rhinologist is "put on the spot." The patient is unwilling, often, to reform his contributing habits and mode of life, and the physician is only too apt to feel that his assistance is unnecessary, since he conceives the ethmoid infection to be the sole cause of the bodily ailment for which he is requesting help and not that the original underlying cause must also receive adequate attention. The general attitude on each side is apt to be on the order of "pull Dick, pull Devil." And I feel that this attitude of the attending physician and the patient is one reason for the sometimes hasty resort to radical procedures, where the time element necessary for careful study and conservative therapeutics, and often restudy with its accompanying expense, is classed

as dilatory. Hasty resort to radical surgery, without due study and consideration, except in emergency cases, which everyone knows exist, contravenes Lewis' philosophy, with which most of us are in accord, that vital tissues should be given a chance for regeneration, with as little permanent destruction of function as possible. Lewis himself, as he has convincingly stated in numerous papers, recognizes surgery as of importance in the general reconstruction process, but decries radical or function destroying surgery except in cases of grave danger to life or the function of important adjacent organs, as being, in the long run, a hindrance rather than a help to the attainment of the general well being of the patient.

The proper management of a case of acute ethmoiditis consists not only in curing it in the shortest possible time, but of conducting studies to see if it is indeed an acute entity, or if it is an acute exacerbation of a chronic, though latent, process, and if the former, to institute measures to prevent the development of chronicity. These measures will probably consist in the correction of such nasal abnormalities as septal deviations which are conducive to ethmoid infection, and especially to chronicity, and to apply any constitutional or systemic measures that are indicated by the careful studies undertaken.

The management of a chronic infection of the ethmoid will naturally vary according to the amount, position and kind of pathology present, whether it is general or localized in one or more cells, whether or not complicated by frontal or maxillary suppuration, and whether previous surgery has been employed. All questions cannot be considered in a short time, so let us discuss the different forms of management that may be used, commenting upon them as we go along.

Conservative nonsurgical measures are usually worth a trial, and sometimes will yield astonishing results where the pathologic condition is not too advanced. In the first place, allergy should be either ruled out or recognized. This can be done by a careful history, a careful examination, and, if indicated, a careful, thorough study for sensitizations. If such are found, desensitization or avoidance of the offending allergens is the obvious course to be pursued. Defects in metabolism should be corrected and vitamins A and D administered. Fresh vegetables and greens and cod liver oil or its equivalent are administered and attention paid to a normally balanced diet. Exercises, active and passive, massage, attention to posture and breathing exercises increase the general well being of the patient and are a definite aid to treatment. Suppuration from other infected sinuses

should be cared for, if possible, since it is well recognized that when frontal or maxillary discharge has been eliminated the ethmoid infection is also likely to clear up. The converse to this proposition is undoubtedly also true.

In the meanwhile, what can be done locally for the comfort of the patient? For the milder cases, much. Baking the ethmoid area with the infra-red lamp is of some value, but the patient must not be turned out into cold air immediately afterwards. Shrinking and lavage are useful measures. The shrinking is usually accomplished by covering the ethmoid region and middle turbinate as nearly as possible with a cotton pack soaked in a mild solution of cocain and ephredrin or weak epinephrin. This is left in place for fifteen or twenty minutes, usually while the baking is taking place. The nose may now be gently douched with warm saline or boric acid solution, and repacked with the tampons so strongly advocated by R. H. Skillern, if the nasal chambers are sufficiently roomy. This pack consists of cotton sufficient to cover the ethmoid portion of the lateral nasal wall, soaked in a 25 to 50 per cent solution of colloid silver in water and glycerin, equal parts. This is preferably introduced hot, and should be kept in place for from two to six hours. Most patients naturally cannot stay around the waiting room for this length of time, so they are allowed to go about their affairs, with a generous supply of soft paper tissue in their pockets to care for emergencies. They return at a given time for the removal of the pack, or if this is not feasible, a strong, black silk thread is looped about it, the loose end tucked into the vestibule and the patient instructed to remove the pack at a specified hour. This all works out well, but Skillern's other dictum is harder to enforce—namely, that these treatments must, to be effective, be repeated at second day intervals for several weeks, the time between being gradually lengthened as the condition improves. Samuel R. Skillern has recently improved upon this technic by incorporating in the mass of the pack a small metal tape to which is attached the positive pole of the diathermy machine, the negative electrode being placed upon the back of the patient's neck. This generates heat in the parts being treated and seems to be very effective in some cases, but it must be used with caution, the current kept low and the time of treatment limited to five up to twenty minutes with the current set at 250 milliamperes. It is well to start with the minimum dosage, and if the patient experiences no discomfort, or as he develops tolerance, the current can be increased to a maximum of 700 milliamperes. Straight medical diathermy also has its advocates. In those cases where the infection is mild in character and principally

confined to the posterior series, where it is difficult or impossible to place a pack with the turbinate in situ, the Proetz displacement irrigation is often most effective. It is a very simple office procedure that patients, even young children, take to readily if they once see the method demonstrated upon another individual, and they often become enthusiastic about the results obtained. Various solutions may be used, the most common being normal saline solution, one-half of 1 per cent ephedrin sulphate in normal saline, or, as Ridpath prefers, poppy seed oil. The method is reasonably safe, although on two occasions I have seen some middle ear inflammation develop, which may or may not have been a coincidence. Because of this I rarely use the method in acute ethmoid infections. According to Proetz, its efficacy depends upon the dilution and liquifaction of the sinus secretions and the keeping of the medicament in contact with the inflamed sinus mucosa for considerable periods of time. I have a few patients who have developed the art of giving themselves their own Proetz irrigations at home.

R. H. Skillern's dictum was that aeration and drainage, with strong emphasis upon the former, were always necessary for the cure of these cases, which is, I believe, the generally accepted opinion today. If hyperplasias can be so reduced by either surgical or non-surgical means, that sufficient aeration of the cavities can take place, the lining mucosa may return to normal. Intranasal applications of radium in hyperplastic cases has been advocated for this purpose by McCullagh and others, but the method has received little popularity. Roentgen ray therapy stands somewhat in the same class, particularly among the radiologists; in some few instances I have thought it was beneficial. Vaccine therapy, both to prevent the annoying acute exacerbations and to attempt to actually cure the sinus infection, has a stronger appeal and has been much more extensively used. Some knowledge of the bacteriology of the nose is needed in order to secure the best results. The culture must be well taken, by the rhinologist, and the vaccine made by an expert. Improperly prepared vaccine is useless, much more so than a commercial stock, which at times appears to give results, but only where aeration and drainage have been secured. Realizing that the bacterial flora of the nose may, and often does, change from time to time, re-cultures should be made and a check kept upon the type of offending organisms. Fenton has been particularly enthusiastic in the results obtained in the treatment of mild, posterior series infections, characterized chiefly by postnasal discharge. He believes the chief offending organism to be one having hemolytic properties, and for its detection it is necessary to implant

and grow the culture upon media containing human blood. In all vaccine therapy, according to Kolmer, the initial dosage should be small, working up gradually to the maximum at seven day intervals for a period of from ten to twenty injections. While striking results are sometimes obtained by more heroic measures, he feels that the cause for failure in these cases is because the patient has, somewhere in the course, been forced into the negative phase. The dosage should at all times be kept below the reaction limit, be it local, focal or general. In spite of this undoubtedly wise policy, I have seen startling improvements follow immediately upon the accidental production of a severe general reaction. Good results have also been reported following the administration of nonspecific protein therapy, such as milk or its commercial substitutes, but in chronic cases I have found this disappointing. In a somewhat similar category can be placed the local application of vaccines, as advocated years ago by Hays and Watson-Williams, or the more modern bacteriophage, which is as yet still too new to be properly evaluated. Ferris Smith is enthusiastic for the latter technic, using it as treatment subsequent to his radical ethmoid operations.

So much for ultra-conservative treatment. In the milder cases much may be thereby accomplished by patience and persistence both on the part of the patient and the doctor, but it has little to offer for the individual who fails to carry out orders with meticulous detail and who reports for treatment at irregular and often widely separated intervals. In children, with marked tonsil and adenoid hypertrophy and sepsis, a preliminary tonsil and adenoid operation, including all hypertrophied or infected lymphoid structures in the pharynx and nasopharynx, is definitely indicated, as taught by Dean, who found that this procedure alone cured a certain percentage of sinus infections, although the studies of Kaiser, among the Rochester (N. Y.) school children, seemed to show, statistically, that the indiscriminate removal of tonsils and adenoids, without proper study and selection, failed to give encouraging results. It is in many cases absolutely necessary to do a preliminary submucous septum resection before good results from either conservative or radical treatment can be hoped for, and this includes reoperation in those cases where an inadequate septum operation has already been done.

When these conservative measures, thoroughly tried out, fail, the next step to be considered is sinus surgery, and of all of the many types suggested the most simple is fracture of the middle turbinate, either away from the lateral nasal wall in the case of disease of the anterior series, or towards the lateral wall if the cells of the posterior

series are involved. The rationale of this is, of course, to obtain increased aeration and drainage, and, if this proves insufficient, part or all of the middle turbinate must be sacrificed. If disease is definitely limited to the bulla and infundibular cells, the removal of the anterior one-third to one-half may suffice, but in the case of posterior involvement or infection of all the cells of the ethmoid labyrinth, the whole middle turbinated body should be cleanly excised, except the extreme posterior tip. By leaving this portion intact it is usually possible to avoid the serious or at least annoying hemorrhage from the branches of the nasopalatine artery, that sometimes accompanies complete removal. Since the middle turbinate is, in the large majority of instances, the chief anatomic cause of the obstruction to aeration and ventilation of the ethmoid cells, its removal seems wise in any surgical effort to restore the physiologic balance. At times nothing more need be done, and in any event the application of the nasal packs and other conservative therapeutic measures have now vastly more chance of success and they should all be given a retrial. There are many who condemn this sacrifice of the middle turbinate, charging that crusting, loss of the sense of smell and uncomfortable nasal dryness follow this procedure, but with this I cannot agree. The mucosa of the middle turbinate is not supplied with cavernous tissue or glands to the same extent as is the inferior turbinate, and olfaction should not be lost if the line of resection corresponds to its junction with the main ethmoid body, since the olfactory nerve terminal filaments do not extend so far downward. In all ethmoid surgery these terminal olfactory filaments in the mucosa of the upper mesial wall of the ethmoid must be respected, not only for the preservation of the sense of smell, if indeed it has not already disappeared, but to avoid the very real danger of the transference of infection to the spaces of the pia-arachnoid through the perineural sheaths, which are really dural prolongations, as was pointed out years ago by Key and Retzius. With the middle turbinate out of the way, some improvement in drainage and aeration will probably take place, and certainly all intranasal therapy will be much more effective.

What is to be done about the polypi when present? Skillern always taught that a certain amount of subsidence can be expected if the case is brought under control by any of the above mentioned measures, especially if the hyperplasias are intracellular and small in size. For those that are extracellular, it is surely wiser to remove them in the customary manner with the snare, and at the same time bite out with forceps the hyperostoses which are frequently the points of attachment. If this is not done, recurrence is probable. This pro-

cedure also helps to establish ventilation and drainage and allows more effective medication. In allergic cases, where the main treatment is desensitization, this rule still holds good, for it is unreasonable to suppose that a large polyp, with a considerable amount of fibrous tissue and a long, tenuous pedicle, would be apt to undergo recession.

One other conservative measure must be mentioned—the employment of mass suction, or negative pressure, to promote drainage and evacuation of cell contents. This has been employed with some success and is particularly applicable to the ethmoid and frontal cells where we already have a certain amount of dependent drainage, due to the location of the ostia. It is obviously of no service in the maxillary and sphenoid sinuses, where the ostia are located near the top of the cavity, unless by posture this condition can be temporarily reversed. In addition to this, it can readily be seen that suction cannot evacuate the fluid content of a noncollapsible cavity, unless a corresponding amount of air is forced in to replace it. If suction is to be employed, therefore, it should be intermittent in character, so that, as a vacuum is formed in the cell, air may be sucked in to restore the balance. The apparatus designed by Coffin does this admirably. The use of negative pressure, however, is not without its dangers, and it should always be employed with extreme caution and with very small force. I have had one case where the patient reported that strong suction had been used, with great resulting pain, and who presented, when I first saw her, undoubted signs of an acutely obstructed frontal sinus. External operation showed that practically the entire sinus lining, chronically diseased and probably loosely attached, had been torn from the bone and lay in a closely packed plug in the upper end of the nasofrontal duct, completely blocking this channel. Suction through a fine tube, such as the Frazier-Grant brain canula, is safe and is a useful adjunct to our cleansing paraphernalia. By its intelligent employment the nasal chambers can be quickly made free of secretion, which can often be thus traced to its source, materially aiding in establishing a diagnosis.

If it seems reasonably certain that the infection is definitely limited to a certain ethmoid cell or group of cells, the attempted drainage of this area is justified, and in such case no more than drainage should be attempted, although it is difficult to resist at times the temptation to open other cells on speculation. Skillern especially warned against this tendency on the part of the operator, and he also believed that disease of the anterior sinuses was almost always complicated by frontal sinus infection. The one cell that offers possibilities of individual drainage with satisfactory results is the bulla ethmoid-

alis, as has been pointed out by Herbert Tilley. Removal of the inner inferior wall of this cell with punch forceps or with the hook is sometimes successful, particularly if there is a closed empyema present.

None of the measures outlined above runs counter to Lewis' postulate that function should be maintained or restored as far as may be, with the possible exception of the middle turbinectomy. Suppose now that all of these measures have failed. Shall we and the patient be content with what relief, if any, has been obtained or shall we advocate further surgery, any form of which must be classed as radical or destructive in type? The answer must depend entirely on the individual, his station in life, the degree of his disability, and the amount of discomfort it is causing. If there is no urgent call for the eradication of a focus of infection, as indicated by systemic disease or danger to adjacent organs, if there is no disabling headache, no bad breath, no serious nasal obstruction, no overwhelming amount of secretion, and the patient is willing and able to continue with conservative treatment for an unlimited period, radical surgery may be deferred. But if one or more of these signs and symptoms is sufficiently pronounced to interfere with the pursuit of his ordinary mode of life, either work or play, the further attempt to a cure should be undertaken. It is difficult to estimate the amount of disability a headache may produce, or indeed to estimate the degree of that headache. What is severe pain to one may not interfere greatly with the work or play of another. Excessive discharge or odor may inconvenience the laborer but little, while in a teacher, a singer, a dentist or a rhinologist it may be fatal to his efforts to make a livelihood. Every case must, therefore, be considered on its own merits. If radical operation is to be undertaken, the type of such operation may well depend upon the degree of the pathology present, as well as the distribution of the ethmoid cells as determined by x-ray. In all cases the operation with the least destruction that will still offer probabilities of accomplishing the desired result should be the one of choice, and we should not confine our ethmoid surgery to any one standard type for all cases. Each type has its own merits.

The most conservative type of radical ethmoid surgery is the intranasal exenteration of the cells, leaving the middle turbinate intact. This is, of course, only possible where this structure has not already been removed. This is the operation practiced by Halle, Pratt and Sullivan. Its safety lies in the fact that the middle turbinate is always present to guard the cribriform plate from injury and the perineural sheaths of the olfactory filaments are not opened. It has a certain amount of danger because one must work in a long tunnel,

for the most part without the aid of vision. It will often drain and ventilate satisfactorily and there is little loss of function resulting from it. Mosher partially employs this method, but at the end removes the pendulous portion of the middle turbinate, leaving intact the dangerous so-called "superior overhang." Skillern followed the Hajek method, working from behind forward with the hook and forceps, while Sluder employed his own knife to remove the ethmoid mass en bloc. Skillern found it advantageous in most cases to remove the unciform process as a preliminary step, thus gaining from ¼ to ⅜ inches more room for instrumentation and for visualizing the operative field. Mosher, after his initial break with the curette into the anterior ethmoid cells, reversed the curette and worked forward to reach and drain the cells of the agger nasi and about the infundibulum. The method employed matters little; the object is to exenterate as completely as possible all diseased or nondiseased cells. When well done it is often effective, although admittedly not without danger, but it is often impossible where there are outlying cells, to reach and drain all of them, and if this is not done discharge and disease and discomfort will not always be entirely relieved.

What remains then for those cases where this radical procedure has failed or where it is, from the beginning, apparent from the condition present that failure is likely to be the result? The Killian operation, if the frontal is also involved; or the external fronto-ethmo-sphenoid operation of Jansen, Lynch, Sewall and Ferris Smith. This operation gives us the best chance for a complete cellular exenteration, with the advantages of good vision, good hemostasis, the option of frontal and sphenoid drainage if desired and insignificant deformity. It is, moreover, by all odds, the safest operation for complete exenteration that has been devised, and by its use frontal bullæ, orbital cells and most other ethmoid extensions may be reached.

The postoperative treatment is of importance. Hemorrhage should not be checked by tight packing, thus damming infected material into newly opened blood and lymph spaces. When bleeding occurs it can usually be controlled by the application of adrenalin or, as I prefer, a mixture of iodin, benzoin and guaiac. For some days following operation no treatment at all should be used except occasionally shrinking of the inferior turbinate for the improvement of ventilation and drainage, and the use of heat furnished by the infrared lamp. The patient should be instructed not to blow the nose and to "hawk" as little as possible. Crusts and secretion should not be disturbed or, as a rule, the nose douched. Later this cleansing may be practiced with safety but not at first. After the first few days crusts

may be removed, as well as tags of tissue missed at the time of operation, which will now be swollen to many times their original insignificant size. At this time the heated colloidal-silver glycerin tampons are resumed, and in the course of a few weeks we may expect, if we are successful, that our cases will be healed.

If an entirely satisfactory result has been obtained we expect to see in time a roomy nose in its upper portion, with the inferior turbinate functioning to prevent excessive dryness, the external and superior boundaries covered with glistening, normal appearing mucous membrane, the sense of smell intact (if it was not previously destroyed), no secretion and no crusting, which is, after all, but dried secretion. In unfavorable cases we find the ethmoid area after a while partially occluded by masses of infected and suppurating granulations or divided into pockets by synechia. Purulent secretion is evident and crusting not uncommon. Such a condition is most discouraging and usually means failure to remove all infected tissue or to drain all infected cells, which is not always possible. Or, instead of granulations, the cavity may be filled with dense cicatricial tissue. Conservative treatment will often ameliorate this condition; if not, reoperation is the other choice. Not all cases that have had radical surgery present this picture, however much some observers would have us believe it to be the usual result. If the work is skillfully done by men of experience who have studied their cases thoroughly and have adopted the proper procedure for the individual, the outcome is eminently satisfactory to both patient and surgeon in a large percentage of cases. Those who see only poor results from radical surgery possibly forget that it is only the unsuccessful case that comes eventually under their care and that the cured cases return to their own surgeons. Possibly the most common result obtained is midway between these extremes, where the patient is relieved of the most distressing symptoms, but where some discharge remains, which may be increased, with headache, when an acute nasal infection develops.

The dangers that may result from an ethmoiditis, acute or chronic, with or without operation, are well known. Meningitis and thrombophlebitis of the cavernous sinus are the worst. In addition orbital cellulitis is a not infrequent complication of the acute infections. The optic nerve may be injured during an operation, but *should never* be so. The orbit may easily be entered during the performance of an intranasal operation, but as a rule, little harm results, a black eye and some orbital pain being the usual sequel. Emphysema occurs at times from nose blowing forcing air into the soft tissues. Osteomyelitis does not result from disease of the ethmoid unaccom-

panied by either frontal or maxillary sinus infection. Temporary diplopia is not very uncommon following external radical operations, where the pulley of the superior oblique muscle has been detached, but fortunately is usually of short duration. Rarely it may be permanent. Tilley has seen a permanent diplopia from this cause but once.

As to methods of repair, the work of Knowleton and McGregor shows that in the maxillary sinus granulations cover the bone from which the mucoperiostium has been removed, and over these spreads a new epithelium which in time becomes ciliated columnar with the proper complement of glandular structures. This should be the usual sequence—where it fails it may be because the underlying bone has been too badly diseased or has been injured by curettage. Hill shows regeneration of the mucosa in the sphenoid sinus, while Ersner and the writer have found it to be true of the frontal. Reasoning by analogy, therefore, we may expect the same thing in the postoperative repair of the exenterated ethmoid mass. It is to avoid injury to the bone that Tilley advocates removing mucosa with forceps of the Luc type, while Sewall and Ferris Smith scrub it off with gauze pledgets soaked in 5 per cent picric acid in 35 per cent acetone solution.

While some allergic patients will recover from their sinus manifestations when the allergy is brought under control, this is not always the case, especially where a suppurative condition has been superimposed. In such cases well designed and executed sinus surgery frequently gives relief from the hay fever and asthma, but operation should not be undertaken during the hay fever season. That the cure of the sinus infection may not be permanent does not detract from the truth of this observation, for the new sinus mucosa may become infected as was the original. If so, we may also look with some confidence for the return of the allergic symptoms.

I would like to be able to give you some sort of percentage of favorable results to be looked for, but this I am unable to do except, possibly, in the case of the combined external fronto-ethmo-sphenoid operation. R. A. Luongo of my Graduate Hospital staff has recently gone over the records of the first fifty cases done there under modern technic, with the following results: The constitutional symptoms were cured in 50 per cent, were improved in 30 per cent, while in 20 per cent there was no improvement; local symptoms cured in 60 per cent, improved in 10 per cent, not improved in 30 per cent.

In conclusion, may I quote from Tilley, Skillern and Lewis. Herbert Tilley: "In any operation which entails opening of the

ethmoid labyrinth it must be borne in mind that its mazes are intricate and when infected by pyogenic organisms the accurate localization of the extent of disease will often tax the diagnostic acumen of the most experienced rhinologist, and its treatment should steady the hand of the boldest operator. He who attacks inflammatory lesions of the ethmoid labyrinth without detailed knowledge of its anatomy and pathology must surely hold very lightly the safety and sanctity of the life entrusted to him."

Eugene R. Lewis: "Conservative surgical procedures co-operating with natural defense and healing processes frequently prove of tremendous value; in critical conditions this may turn the tide, averting a fatal outcome, by timely venting of purulent accumulations. I am a staunch advocate of nasal surgery, provided it is conceived logically and executed conservatively. It is my well considered opinion that patients as a whole would be better off by complete abandonment of radical sinus surgery."

Ross H. Skillern: "Let me state that I firmly believe, as time goes on, we will no more look upon our treatments in the light of a hope rather than a faith, but will finally agree upon a standard method of handling these cases, with such uniformly happy results that the subject will no longer be termed 'The Ethmoid Problem'."

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V.

REPORT ON TEN CASES OF SUPPURATION IN THE PETROSAL PYRAMID.*

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Since we last reported on suppuration of the petrosal pyramid many additional papers have appeared in the literature, dealing with the clinical and the surgical aspects of the problem. Many of these reports have tended to increase confusion. Some were based upon a paucity of cases which rendered a serious discussion of the conclusions unwarranted, because the experiences upon which they were based were so limited. In other instances, cases were reported to illustrate points in contention which, when they were critically analyzed, hardly met the fundamentals necessary to establish them as unquestionable cases of petrosal suppuration. Since these cases were diagnostically equivocal, the validity of the conclusions must be dubious.

In addition there are still too many cases of terminal meningitis reported; in these the necropsy findings demonstrated that during the course of the evolution of the lesion in the cranial cavity a suppuration in the petrosal pyramid had been present. The clinical implications of the lesion, while still confined within the anatomic limits of the pepetrosa, were entirely overlooked during life. In these cases the report is predicated upon the postmortem findings, and this has led to a failure, in many instances, to separate the clinical picture arising from the petrosal suppuration from that presented by the invasion of the meninges or the brain.

Where an intracranial lesion develops, either a brain abscess or an intracranial extension of a cholesteatoma, and death follows, there exist in the tissues secondary and unimportant local reactions beside the obvious causes of death; these secondary reactions have led some to attach a special significance to them. Some have interpreted these reactionary inflammations as evidence of chronic bone lesion within

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the petrosal pyramid. Except for chronic osteomyelitis, syphilis or tuberculosis, we know of no chronic lesion which has been pathologically demonstrable within the petrosal pyramid. What we have observed has been a tract of purulency leading from the tympanomastoid cavity to various portions of the pyramid. This tract, persisting for a considerable period of time, has permitted a slow drainage from the depths of the petrosa and thus has led to a chronic otorrhea or a chronic discharge from the mastoid wound. It is merely an endeavor of nature to evacuate a collection of pus, the formation of which was always associated with an acute suppurative tympanomastoiditis. In these instances the fact that drainage had not been impeded has resulted in an absence of the clinical signs which are ordinarily associated with the acute and subacute cases of suppurative petrositis. However, at this time we shall not further discuss this mooted question in bone pathology.

The literature also presents differing opinions anent the condition of the structures within the tympanic cavity in the presence of a diagnosed suppuration of the petrosal pyramid. With the arguments which are advanced to advocate limiting of surgery to a simple mastoidectomy we have little sympathy. The pleas for so-called conservatism are not scientifically grounded. Each type of lesion in the petrosal pyramid in its group presents a given specific problem and a definite set of indications for surgical therapy. We have never contended that one method of surgical approach to these problems covered all cases. Clinical experience has taught us, however, that failure to meet each specific set of indications which a given case presents is not necessarily desirable conservatism. Were it possible to study some of the fatal cases before death occurred, then it would perhaps be possible to point out at which stage in the case history protracted observation and inaction or restricted surgery was responsible for the failure to obtain a cure.

The hazards of conservatism have not yet been critically weighed. In some of the published reports where conservatism has apparently been successful, the diagnosis of a suppurative lesion in the petrosa is not entirely conclusive.

There is also noted in the current literature numerous articles detailing particulars of surgical technic and many so-called new and improved methods of surgical attack. It would hardly be fair to analyze critically comparative results for the number of cases submitted to the "improved" technic is very meager. In addition, those reporting improved technics have failed to evidence sufficient surgical

experience with the fundamental procedures which have had the support of competent authorities both here and abroad. Consequently these observers are not in a position to make a comparison of methods and results.

Finally, the literature contains reports concerned with basic studies. The conclusions based on microscopic findings of the pathologic material are impressive. While such work has clarified mooted academic questions, it presents nothing to cause us to change our standpoint regarding the fundamentals of diagnosis and of surgical therapy which we detailed in our first communication.[1]

In making our clinical report, let us stress our position regarding diagnosis. We feel that the lesion cannot be detected by the roentgen findings alone; nor can a diagnosis be considered authoritive upon clinical data alone. The two factors must be taken together. It is also necessary that all other factors which conceivably could cause the symptom complex must be checked and eliminated. Finally, to become acceptable as a proven case with an unquestionable diagnosis, the operative findings or the autopsy must substantiate the presence of the lesion.

Since our last report, either separately or together, we have studied fifteen additional cases, ten of which comprise this report. Of the five cases not included here, four were seen and operated on in consultations with surgeons not in New York City. These four cases were worked up, diagnosed and handled postoperatively by the surgeons in attendance upon them and the cases are theirs to report. One case which we handled and which was proven and operated upon by us and which recovered, cannot here be reported because in compiling the case history the record was found so faulty that we were unable to include it in this presentation. We comment upon it for statistical purposes only. This report is limited strictly to the cases which we have studied in minute detail and have ourselves handled throughout the illness. In the running comment at the end of each case will be found the basis of those fundamentals which we have from time to time published and which these cases further substantiate.

REPORT OF CASES.

CASE 1.—C. S., female, 12 years old, was first seen by us at Beth Israel Hospital on February 3, 1934. We obtained the following history from Dr. M. J. Stearns of Ogdensburg. He first saw the patient on February 2nd. The child had scarlet fever four weeks previously. There was a right acute purulent otitis which developed one week after the onset of the scarlatina. There was a profuse otorrhea which lasted a few days but which reappeared after a short period. There was

occasional pain over the right eye which lasted for a short time. On February 1st, the child had an attack of severe pain in the head and vomited. On the day of her examination, there was marked rigidity of the neck and projectile vomiting, but the headache was not severe. The spinal fluid was cloudy and under pressure; it contained 1089 cells, 90 per cent of which were polynuclears. Copper reduction was present but reduced in amount, globulin was increased. There were no organisms to be found on smear. Roentgen examination of the mastoid showed a large cellular process with extensive pneumatization, moderate cloudiness but no apparent breaking down of its cell walls. The petrosa on a base plate showed definite obliteration of the cell structure on the right side, with beginning rarefaction at the tip.

Our examination just prior to operation on February 3rd corroborated the above findings; and, in addition, we found a vertical nystagmus present. A simple mastoidectomy was first performed; we found some pus in the tip and postsinal cells which communicated with the cells in the digastric groove. The remainder of the mastoid cells were intact but the bony septa were softer than normal and appeared grossly diseased. The wound was then converted into a radical cavity and the tympanic cavity was found filled with granulations. After a complete toilet of the middle ear space, the anterior buttress was thinned out, bringing the orifice of the eustachian tube into full view. No evidence of a fistula was found in the epitympanic space, antrum or peribulbar area. At the mouth of the eustachian tube, however, at the area we select for making our entrance to the petrous tip, a fistula was found which, when probed, led to the petrous tip. This consisted of one large cavity; and probing about its boundaries elicited the fact that there was a dehiscence of the superior surface of the petrosa. The entire inner table of the mastoid process was next removed exposing the dura and lateral sinus. The sinus was inadvertently torn during this procedure and the hemorrhage was controlled with iodoform plugs. Lipiodol was next injected into the fistula at the tubal orifice. This procedure was followed by an escape of about two teaspoonsful of thick yellow pus which oozed out of the osseous tissue between the superior and the horizontal semicircular canals. The tract from which the pus was forced out could not be grossly demonstrated nor visualized with the naked eye. The wound was left open for observation.

Roentgen examination following the lipiodol injection showed the chemical to be distributed around the petrous areas into the cranial cavity on both sides, spreading under the tentorium, around the pons and into the foramen magnum.

The child was given 100 cc. of whole blood intravenously on February 3rd, 75 cc. on the 5th, and 50 cc. on the 8th. On February 4th the cervical rigidity was less marked. The Babinski, Kernig and vertical nystagmus were still present. By the 7th all neurologic signs had disappeared. The plugs were removed from the injured sinus on the 11th. The culture of the mastoid tissue showed a nonhemolytic diphtheroid bacilli. The temperature remained around 100 with occasional rises to 101 and 102 throughout the entire postoperative period. All eye pain disappeared immediately after operation. The plastic operation on the membranous canal wall was performed on February 21st, and the child was discharged from the hospital on March 3rd.

The patient, heard from in October, 1934, is well and her ear is dry.

Comment. This is the first case we have seen wherein a suppurative lesion of the petrous apex was evident both clinically and roent-

Fig. 1. Case 1.—Base plate showing distribution of lipiodol in cranial cavity.

genologically prior to the performance of the original surgery on the mastoid process. At operation very little pathology was found within the mastoid proper; whereas, an empyema of the apex which had already perforated and given rise to meningeal reaction was already present before operation. This bears out our view that a petrous suppuration is a complication of the otitis media and is not a complication of the mastoid infection.

Furthermore, it demonstrates that it is possible sometimes to effect a cure in a case wherein rupture of the superior petrosal surface has already occurred, by simple drainage and enlargement of the fistula. Naturally, the sooner such evacuation is accomplished, the better the prognosis.

This case also shows the imperative need surgically to expose the middle ear when a fistula is not found at the simple mastoidectomy. While a fistulous opening did exist in the epitympanic space in this case, it was not visible to the naked eye and it would have been overlooked had it not been for the sight of pus oozing through it when lipiodol was injected through the fistulous opening found at the mouth of the eustachian tube.

To have stopped the operative procedure at the termination of the simple mastoidectomy would have meant missing the tympanic fistula entirely. Several of the cases in this series show the presence of multiple fistulæ and, in addition, two cases of ours which are not reported in this series also presented multiple fistulæ. In the one of these two cases drainage of the fistula in the region of the antrum, behind the superior semicircular canal, did not suffice to effect a cure; but the subsequent performance of a radical mastoidectomy by us revealed the presence of the second fistula at the mouth of the eustachian tube. Drainage of this, followed by washing through from one tract to the other, cleared the case and resulted in a cure.

Our findings in these cases confirm Lange's[2] original observation that the peritubal cells are usually the avenue of escape for the pus, whereas the perilabyrinthine cells emanating from the epitympanic space around the superior canal are the avenue for its entrance into the petrosa. Consequently the rationale of our procedure for the closed empyema of the apex is again substantiated, as it affords drainage at its most advantageous point, and at a place where nature spontaneously seeks to evacuate it.

What would have been gained in this case by performing a subdural operation? The lesion was attacked and cured by the drain-

Fig. 2. Case 1.—Side view. Lipiodol in posterior fossa and through foramen magnum.

age afforded through widening the fistula and subsequent lavage. The drainage was from the most dependent portion of the pus collection and therefore gravity helped evacuation. To have exposed the tip and its dehiscence through any one of the subdural approaches would not have effected ideal drainage, because a cavity in the petrosa would have been left underneath the dehiscence, and this would continue to act as a reservoir for the pus, and only the overflow would escape upward and appear subdurally. Further comments on other cases of this series stress more in regard to the subdural method of approach.

Finally, it is to be noted that the endocranial cavity was apparently sterile. The abscess formation had localized itself. The trauma incidental to separation of the dura from the bone would have invited subsequent infection and development of a meningeal lesion, the prevention of which was our main concern.

CASE 2.—L. L., female, 6 years old, was seen by us on June 2, 1934, following an upper respiratory infection. Nine weeks before we saw her she developed a left acute otitis media with a spontaneous rupture of the membrana tympani. The temperature ranged between 99 and 103 for two and a half weeks and then the patient underwent a simple mastoidectomy performed by Dr. Culkin. Following the operation, the child complained of pain in the midfrontal area. The right membrana tympani was opened when, during the postoperative period, the temperature reached 104. Following this paracentesis the temperature dropped to 101 and remained at about that level until one week prior to coming under our observation when it had returned to normal. Within the past three days, however, the temperature again rose to 102 and the child complained of headaches.

Examination revealed the left middle ear to be full of pus coming out under pressure. The right drum was reddened but intact. Neurologic examination showed no abnormality. Roentgenogram of petrosa showed a suppurative lesion of the left petrous apex.

The patient was admitted to the Beth Israel Hospital on June 4th. Temperature on admission was 101. Blood count: R. B. C. 4,190,000; hemoglobin 72 per cent; W. B. C. 10,500; polynuclears 82 segmented, 3 staffs.

The child was operated upon the same day. The mastoid wound was reopened and the granulations removed. The sinus and dura were found exposed by the previous surgical procedure. A search for a fistula was made in the epitympanic space and in the peribulbar area, but none was found. The wound was converted into a radical cavity and the middle ear was found filled with granulations. A complete toilet of the tympanic cavity was performed and the orifice of the eustachian tube brought into full view by removal of the anterior buttress. A fistula was found at the mouth of the tube which passed forward and inward into a cavity filled with granulations. This could be determined by probing. The tract and the cavity were curetted with a Yankauer curette. The wound was lightly packed with rubber dam and left open.

On the following day the child complained of continuous pain over the left eye. A full dressing was performed. Pus was found coming out under pressure

from the fistula at the tubal orifice. About three teaspoonsful were recovered. A needle was inserted into the fistula and by means of a syringe attached to it, air was forcibly blown into the petrosa. This was followed by an oozing of pus from a microscopic fistulous opening located anterior to the vertical semicircular canal. The cavity was then washed with saline, followed by air injection. The minute fistulous opening was widened with a fine rasp and a rubber dam drain inserted into it.

On June 6th the temperature rose to 102.6, but from that time on dropped by lysis to reach normal limits by June 15th. Daily dressings and irrigation of the fistulous openings into the petrosa were continued until June 22nd, when no more pus could be recovered. Following a dressing on June 14th, a slight facial weakness was noted which lasted for about six days and completely disappeared. The plastic on the posterior membranous canal wall and the closure of the wound was performed on June 25th and the child was discharged from the hospital on June 27th. At the present writing she is completely cured.

Comment. Here is presented a case wherein there were two fistulous tracts into the petrosal pyramid. The one located anterior to the vertical semicircular canal was not visible to the naked eye. The thorough exposure of the tympanic cavity was made necessary so that the opening at the mouth of the tube would not be missed. Had we been content to cease our surgery at the simple mastoidectomy with the widening of the epitympanic space, the story of this case would have been different, even had it been possible to visualize this small fistula.

The diagnosis in this case was substantiated by the roentgen plate. The operation for its cure was in reality nothing more than a radical mastoidectomy coupled with the establishment of adequate drainage of the fistula at the mouth of the eustachian tube. The opening found was at the point of selection for entrance to the apex as described by Almour. We desire to stress the fact that in this, as well as in the preceding case, the authors did not perform the operative procedure which they themselves described. It is just such cases which compel us to request those interested to again read our "Indications for the Type of Surgery Required," as given in our original communication.[3] We reiterate that there is no *one* method of treatment for *all* cases of petrous suppuration. The subdural route, in our opinion, is only justified where a subdural collection of pus definitely can be diagnosed beforehand.

In a series of case reports we can hardly digress to treat this topic with the detail it requires. At this time, suffice for us to state that we believe the opening into a sterile endocranium with its absolutely unavoidable trauma to vascular channels passing from the bone to the outer layers of the meninges, when done in the presence of active infection within the petrosal bone tissue, constitutes a dangerous sur-

gical hazard. The rôle this hazard plays in the development of meningeal infection subsequent to the operative procedure is not generally comprehended. Where a subdural collection of pus is present prior to the surgical approach to the subdural space this hazard is materially lessened because of the walling off that accompanies and precedes the formation of the local subdural pus pocket.

The technics of Almour and Ramadier are applicable and should be performed only in the presence of an encapsulated empyema of the apex. Frenckner's technic is to be advocated *only* when a fistulous tract is present through the arch of the superior semicircular canal. In any event, every case of petrous suppuration, regardless of its type, is entitled to a thorough exploration of both the mastoidal and the tympanic portions of the temporal bone to search for the presence of a fistulous opening into the petrosa before any extensive operative procedures are begun. That is why we are not in accord with those who advocate any of the procedures which attempt to avoid a radical exposure of the tympanomastoid area but expose the apex of the petrosa subdurally. To leave the middle ear unopened frequently may result in failure to uncover a fistula at the tubal orifice, the simple drainage of which would suffice to effect a cure and obviate further surgery to the petrosa. Likewise, it would obviate the inherent dangers which opening the cranial cavity entails.

Let us also consider here, because it is germaine to the discussion of this case, further details of the rationale of the subdural approach. The complications of a suppurative lesion of the petrosa occur because a rupture through its boundaries into the cranial cavity has taken place. If a rupture has occurred through the superior surface with the formation of a subdural abscess, this route of approach is then logical and understandable because it reaches the complication of the petrous suppuration. But where the pus is still confined to anatomic limits of the petrous bone the subdural approach makes an artificial opening with the hazards to which we have alluded above.

Even when every subdural method thus far described is effective in reaching the apex without endangering the meninges, this procedure nevertheless immediately creates a subdural infection with its attendant dangers. The operative technics which limit themselves to the confines of the temporal bone, however, tend, as far as possible, to keep the suppurative process within the temporal bone. This constitutes the one objection that we have to the operation of Ramadier, since in his technic an avenue for spread is afforded by the exposure of the carotid canal.

Why are otologists careful to preserve, as far as possible, the anatomic bony limits of the mastoid process in the performance of a simple mastoidectomy? Because the infection is limited to the mastoid process. When are the bony limits of the mastoid process removed? Only when a complication is presented or suspected. Cannot this logic be applied to the types of surgery advocated for a suppuration of another portion of the same bone, bounded by the same dura, the same venous sinuses, and the same meninges and brain? We believe that when the dangers of the subdural approach with its ruthless rupture of blood vessels and its trauma to the dura and subsequent infection are better comprehended, then otologic surgeons will be less prone to employ this route when other and more direct methods of approaching the lesion are available, particularly so when these other avenues of surgical approach carry no such risks, and when cases, as here reported, recover without opening the endocranial cavity.

CASE 3.—S. R., male, 6 years old, contracted measles in March, 1933, during which illness he suddenly experienced pain in the right ear following forceful blowing of his nose. A myringotomy was performed and a profuse otorrhea ensued. Pain and fever then subsided and the child was well for a period of two weeks until on March 28th the temperature rose to 104.5 and the aural discharge became serosanguinous. The fever persisted and the child complained of some generalized headache.

The child was first seen by one of us on April 3, 1933. Otoscopic examination revealed a profuse otorrhea coming away under pressure and a marked drooping of the superior canal wall; definite periostitis and tenderness over the right mastoid process were present. Tonsils and adenoids had been removed. Transillumination of nasal accessory sinuses showed them clear. General physical examination was negative.

The patient was admitted to Beth Israel Hospital on April 3rd. On admission the temperature was 101.2. The urine showed a faint trace of albumin and occasional white and red cells. The blood count showed: R. B. C. 4,270,000; hemoglobin 68 per cent; W. B. C. 13,600; polynuclears 72 segmented, 6 staff cells; mononuclears 22.

A right simple mastoidectomy was performed on April 3rd. After separation of the periosteum, the cortex overlying the region of the lateral sinus was found to be discolored and gangrenous. Upon removal of the cortex, pus gushed out under pressure and subsequently yielded a streptococcus hemolyticus pyogenes on differential culture. The mastoid process was completely coalesced and filled with pus and granulations. The inner table was exposed throughout by complete surgical exenteration of the mastoid contents revealing a mastoid process which had been extensively pneumatized. The wound was closed with interrupted silk sutures and drained through the lower angle with rubber dam.

The temperature rose to 103.2 following operation and for the next two days ranged between 100 and 102.8. On April 4th the nurse noted occasional twitching of the right side of the neck; on the following day sudden jerks of the entire body while asleep and twitchings of the right shoulder and lower extremities. On

April 5th he vomited his lunch and felt drowsy most of the day. General and neurologic examination were entirely negative. The wound was dressed and found to be in excellent condition; the middle ear was dry and the drum healed; three upper sutures were removed. On the 6th and 7th of April the fever ranged between 100 and 101 and the child appeared brighter and more alert. He again vomited his lunch on the 7th. The temperature fell to 99 on April 8th, only to again rise to 101.4 the next day. Until his discharge from the hospital on April 13th the highest temperature was 100.6 and on discharge was 99.4. All sutures and drain were removed from the operative wound and the patient's general condition being good, he was sent home for subsequent ambulatory treatment.

On the day of his return home, the mother noticed that the child squinted with his right eye. The child, when questioned, stated that he had a pain in the right eyeball. A dressing was performed on April 14th and a mild otorrhea was found in right middle ear; this was not under pressure. The mastoid wound was discharging a moderate amount of pus. For the following week the temperature ranged between 99 and 101. There was a constantly increasing severity of the nocturnal retro-orbital pain. Upon the insistence of the parents an ophthalmologist was consulted who found no pathology in the eye.

A definite diagnosis of empyema of the petrous apex was made and the patient readmitted to Beth Israel Hospital on April 20, 1933. On admission the temperature was 100.6. There was a slight swelling over the zygomatic ridge on the right side; there was an equivocal Kernig; neurologic examination otherwise negative; no abducens palsy. Roentgenologic examination of the petrosae with the aid of the base plate and Stenver's position showed the following: Examination of the base of the skull shows the left petrous bone to be pneumatized to the tip. On the right side the anterior portion of the tip shows a definite halisteresis and bone absorption. This process affects the distal third of the pyramid. There is also a slight increase in general intracranial pressure.

The patient was reoperated upon on April 20th. The old incision was reopened. It was found that the swelling over the zygoma was due to a stitch abscess. Careful exploration of the zygoma itself failed to reveal any pathology. The newly formed granulations were removed from the mastoid cavity and zygoma. The antrum was carefully curetted and all granulations removed therefrom. The inner zygomatic wall and outer attic wall were next removed, leaving the ossicles and drum intact. Careful search in this area revealed a fistulous opening situated behind the superior semicircular canal. A probe inserted into this opening took a direction inward, forward and downward. Careful manipulation with this probe was followed by an immediate gush of one teaspoonful of thick creamy pus. This opening was next enlarged with a burr and a Yankauer curette inserted into the fistula and the entire cavity curetted. Soft, mushy granulations were thus removed. One cc. of lipiodol was injected into the cavity. The wound was left open and packed with gauze.

At 7:15 p. m., following operation, the child had a chill which lasted 15 minutes, and the temperature rose to 106.2. Examination of chest negative. Suggestion of right Kernig. At 10 p. m. temperature dropped to 104.4. The wound was dressed and the gauze packing replaced by rubber dam.

On the following day the temperature had dropped to 99.8. Blood count showed R. B. C. 4,180,000; hemoglobin 63 per cent; W. B. C. 11,200; polynuclears 74 segmented, 5 staff cells; mononuclears 23. A transfusion of 75 cc. whole blood

was given by the direct method following which the temperature rose to 102.6. The child still complained of pain in the right eye.

From then on the temperature dropped to normal and remained so until date of discharge from hospital. No further complaint of any kind from the 22nd of April onward. On April 22nd, a dressing was done, the fistula was reprobed and a thin sliver of rubber dam inserted into it. On April 24th the middle ear was dry. On April 30th the fistula was found dry; the drain was removed from it and a probe encountered a bed of granulation tissue. On May 1st the boy complained of pain in the lower jaw which was found to be due to a carious left lower molar. This was extracted. On May 4th the fistula into the apex was found entirely healed and closed over by healthy granulations. The patient was discharged from the hospital and from then on made an uneventful recovery.

Comment. A cure of the petrous suppuration was effected without the performance of a radical mastoidectomy. See Case 5 for fuller comment. The child *has not* perfect hearing despite the fact that nothing was done to the middle ear.

CASE 4.—H. L., male, 19 years old, was admitted to the Beth Israel Hospital on April 22, 1933, with the following history. He acquired an acute upper respiratory infection in February of that year followed by pain in the right cheek and right ear. The right drum ruptured spontaneously and drained profusely. On February 24th a right simple mastoidectomy was performed by Dr. M. L. Harris. For the next four weeks the patient felt well. For the two weeks before admission he has complained of right sided headache and pain in the right eyeball. The temperature range during this time was between 100 and 101. He had a few dizzy spells and on the day before admission vomited. Roentgen examination by Dr. H. K. Taylor showed a diminution in aeration in the basal portion of the right petrous pyramid. The right petrous apex showed a loss of trabeculations, a diminution in aeration and considerable decalcification.

Examination on the date of admission revealed a profuse otorrhea and a considerable amount of discharge coming from the tympanic antrum through the mastoid wound. There was a horizontal nystagmus to the left. Blood count on admission showed R. B. C. 4,850,000; hemoglobin 73 per cent; W. B. C. 16,600; polynuclears 76 segmented, 1 eosinophile, 7 staffs.

On April 23rd the patient was reoperated. The old mastoid wound was reopened and a wider exposure of the zygoma was made. The root of the zygoma contained a number of normal cells; these were cleaned out. A wide exposure of the attic and epitympanic space was accomplished by extensive removal of the inner zygomatic table and outer attic wall. A large fistula was found situated in the quadrant between the horizontal and superior semi-circular canal. This fistula, when probed, took a direction directly inward toward the petrous tip for a distance of 2½ inches. The mouth of the fistula was widened and with the aid of a burr the tract was enlarged to allow a curette to be passed into it. A thorough curettage of the cavity was performed.

On the following day the patient was dressed. The fistula was located and probed. The probing of the apex was followed by momentary sticking pain in the right eye. The fistula was washed out. The headache and deep seated eye pain had disappeared.

For the next seven days the temperature, at first up to 102, gradually dropped to 100. On April 29th he complained of soreness in the throat on the right side. On April 30th pain in the right side of the neck and at the angle of the jaw. On the afternoon of the latter date a slight right external rectus palsy appeared and there was a definite swelling of the right lateral pharyngeal wall.

On May 1st the tongue deviated slightly to the right, indicating interference with the function of the right hypoglossal. On this date, under topical application of cocain, the swelling of the right lateral pharyngeal wall was aspirated and pus obtained. The needle puncture wound continued to ooze pus and was widened with a hemostat inserted and spread. Considerable pus escaped. A probe inserted through this opening followed a direction upward and backward.

On May 3rd the diplopia and external rectus palsy had cleared and the hypoglossal palsy improved. On May 10th all signs had disappeared and the patient was permitted to go home.

Convalescence was uneventful except for a persistent postauricular fistula which was subsequently closed on July 6, 1934.

Comment. It is important to note the time of appearance of the abducens palsy in this case. It occurred on the eighth postoperative day, and its appearance was simultaneous with the discovery of a dissecting abscess which finally presented itself on the lateral pharyngeal wall. It disappeared two days after the abscess was evacuated. This is definite evidence that the sixth nerve palsy was due to an extra-petrosal lesion and supports the prevailing opinion that the appearance of an abducens palsy in the presence of an otitic suppuration is more often than not the result of meningeal irritation and not the accompanying symptom of a petrous suppuration, an opinion which is gaining ground in competent quarters both here and abroad.

CASE 5.—M. C., female, 15 years old, was first seen on March 20, 1933. For the three weeks previously she had had a sore throat, coryza and cough. On March 18th she experienced severe pain in the right ear and a purulent otorrhea appeared. She had been nauseous for three days and had vomited once. Temperature ranged from 100 to 103. She was admitted to the Beth Israel Hospital on March 20th for observation. For the first four days the temperature ranged between 99.2 and 102.2; for the next two days from 99 to 101; it reached normal on March 30th and remained so until she was discharged from the hospital on April 7th.

Roentgen examination of mastoid processes and nasal accessory sinuses on March 21st showed: Marked diminution in aeration of the right mastoid, especially near its tip, and there is some impairment of detail in this region. These findings probably indicate beginning softening. Both mastoids are highly developed pneumatic bones with a well developed zygomatic extension on each side. The petrous bones are pneumatized down to the apices. They show no pathologic changes. Re-examination on March 28th showed no change in the condition as compared to the previous examination. On April 5th the roentgen appearance of the mastoids was practically the same. The plates of the paranasal sinuses showed a right pansinusitis present. During all this time otorrhea persisted.

Despite the persistence of the aural discharge, it appeared as if the lesion in the ear might resolve and, since there was no indication, either on otoscopic examination or on the roentgen plates, for immediate surgical intervention, the patient was discharged from the hospital for further treatment at the office.

Treatment for the right acute pansinusitis was instituted and right antral lavage yielded considerable pus on several washings. On April 10th patient complained of severe headache and pain over the forehead and cheek. An acute coalescent mastoiditis was diagnosed. She was readmitted to the Beth Israel Hospital that day; her temperature was 103. On the following day a right simple mastoidectomy was performed. Upon removal of the cortex, pus came out under pressure. The tip was entirely coalesced and filled with fluid pus and granulations. The remainder of the mastoid cells were filled with pus but the intercellular septa were intact. The mastoid process was extensively pneumatized. The inner table was exposed throughout. A small perisinal abscess was found midway between the knee and the bulb. The outer wall of the sinus was covered with granulations. Further exposure revealed normal sinus wall. There was an extensive development of cells leading from the tip inward toward the peribulbar region. The wound was closed with interrupted sutures and drained with rubber dam.

Her temperature ranged between 100 and 103 until April 13th. Following removal of the stitches on April 13th, a wound infection set in and the postauricular incision broke down completely. Wet dressings were applied and the temperature gradually subsided to between 99 and 100. The right antrum was washed daily and considerable pus obtained each time. All acute pain had disappeared until April 21st, when she began to complain of slight headache. This increased in severity and by the 23rd was definitely supraorbital in character. On dressing the patient that day a fistulous tract was discovered leading inward toward the petrosa and located in the vicinity of the antrum. Probing this tract was followed by a flow of pus. The tract was washed with sterile saline solution.

While a suppurative lesion of the petrous apex was evident, it was thought advisable to first rule out an acute frontal sinusitis. Roentgen examination showed the frontal sinus to be large, multilocular, normally aerated and of about even size. The right antrum still showed involvement. Examination of the petrous pyramids showed the tip of the right pyramid presenting definite osteoporosis with a loss of cellular detail. The cellular illumination was completely absent.

On April 25th the headache was severe. The blood count showed R. B. C. 3,690,000, hemoglobin 55 per cent, W. B. C. 11,300, polynuclears 78 segmented, 3 staff cells. She was reoperated on the following day.

The inner zygomatic wall and a portion of the outer attic wall were removed, bringing the aditus into view. The fistulous opening previously located was identified, enlarged and curetted. A probe went directly forward and inward for 1½ inches into the petrous apex. The wound was left open and packed with iodoform gauze.

The supraorbital pain continued until May 4th and then subsided. Pain over the right cheek was always present and was relieved by frequent antral irrigations. The temperature, except for a few slight rises to 101, remained below 100 and for three days prior to discharge on May 12th was 99.2. By May 11th the right antrum was clear and the patient from then on had an uneventful convalescence.

Comment. This case and the two preceding ones are proven cases of suppuration of the petrous apex wherein a cure was effected by the

drainage of a fistula which was discovered in the surgical exposure afforded by a simple mastoidectomy. One such case has been previously reported by us.

The accidental discovery of the fistula into the petrosa during a dressing led to the correct diagnosis in the presence of the severe acute pansinusitis. The case, in itself alone, merely emphasizes the importance of excluding a nasal accessory sinus involvement as the cause of retro- and supra-orbital eye pain.

A cure was obtained in all three cases. Simple mastoidectomy only was performed in all three, and a fistula was found in all three in the area located above the horizontal and behind the superior semicircular canals. These three cases, although alive and cured, are a disappointment to us. We attempted to follow the prevalent otologic opinion—an opinion which differed from ours—on the advisability of opening the tympanic cavity and sacrificing what could be saved of the hearing of the patient, and at the same time preserve life.

One of these cases is now an epileptic; another suffers from a maniac depressive insanity (we still feel that it may be a frontal lobe abscess); and in the third, the postoperative treatment resulted not only in an ugly scar, but also in a decided diminution in hearing. The third case, at the present writing is apparently in good health despite a great loss in hearing acuity. The other two cases, in addition to their other complaints, also have a marked impairment in hearing. In one instance this factor was sufficient to exclude the patient from favorable action on his application for a license to become a teacher.

Was failure to perform a radical mastoidectomy the responsible factor? We cannot, at this stage of our problem, say with any degree of certainty. Yet let us study Case 4 for a moment. A dissecting lateral pharyngeal abscess presented itself after all subjective complaints had disappeared. Had we performed a radical mastoidectomy at the time of the original search for the petrous focus, would it not have been probable that we may have found a point of disease at the tubal orifice, the treatment of which might have obviated the subsequent appearance of the pharyngeal suppuration and its risks?

Let us view these three cases in the light of the advisability of avoiding a radical mastoidectomy. All three have poor, if not to say bad, hearing. Two of the three (the only ones we have) are sufferers of an added intracranial disturbance (organic or inorganic?). We can not make this clearer because there is no additoinal diagnostic data available.

All these cases, although surgery was limited to the performance of simple mastoidectomy, have very poor hearing. If one re-reads our original reports, it will be found that we stressed the finding in the middle ear, in our proven cases, of hyperplastic thickening of the mucous lining, granulation formations and purulent detritus. In these cases there is also, during the active phase, an active perilabyrinthine osseous reaction which necessarily must influence the intralabyrinthine fluids. Edema of the endo- and peri-lymphatic spaces is also conceivable. While no radical was performed, our hearing tests showed a pronounced loss. We have therefore come to the conclusion, from the clinical material available to us at this time, that in cases of real involvement of the petrosal pyramid in a suppurative lesion, there is little saved by the conservative idea of limiting surgery to a simple mastoidectomy. We save a damaged middle ear which recovered with subsequent contractures of connective tissue, impeding hearing acuity, and at the same time we run the risk of overlooking salient lesions which cannot be brought to view unless the tympanic spaces are opened to inspection and search.

In view of the efforts of the otologic profession to conserve hearing acuity, and in the developmental phase in which this topic stands at the present writing, it was necessary that we should proceed, by trial and error, to arrive at a better comprehension of the terminal results, where conservatism was allowed to influence surgical judgment. With the experience gained by these cases, we are disinclined to continue preserving structures which experience has shown did not function normally subsequent to the development of the intrapetrosal lesion. Whether the neurologic symptoms which these cases additionally present are the result of a low grade encephalitis cannot be said; but this fact stands out: in none of the cases wherein radical mastoidectomy and prompt evisceration of the lesion in the petrosa were performed did we have any such symptoms subsequently.

CASE 6.—F. A. D., male, 10 years old, was admitted to the Beth Israel Hospital on April 11, 1934. During the fall of 1933 he had suffered from a low grade sinus infection. This continued until the 14th of January, 1934, when the child developed an acute bronchitis followed by a bilateral acute purulent otitis for which a myringotomy was performed on January 23rd.

This was followed by pneumonia. On February 8, 1934, a right mastoidectomy was performed. The patient progressed satisfactorily until February 19th, when scarlet fever set in. The progress of the case from then on was uneventful until March 8th, when a swelling appeared over the left mastoid process. A left simple mastoidectomy was then performed.

On March 27th there was a sudden appearance of nausea and of right otalgia. This was followed by the reappearance of a profuse yellow, tenacious discharge

from the right ear. There were pulsations noted in the region of the right antrum. The culture of this discharge yielded a streptococcus hemolyticus. The child then began to complain of severe headaches in the temporoparietal region which were stabbing in character.

On April 4th the right mastoid was reopened. The headaches were relieved following revision. Since then, however, the headaches had returned and there was also a complaint of pain in and about the right eye, very severe, occurring about four times daily. There had been a photophobia for the week prior to admission. The thick right aural discharge had lessened in quantity and had appeared to lessen in amount during the 36 hours prior to our seeing her. There had been a low grade fever throughout the course of the illness. There had been a suggestive weakness of the right external rectus.

On admission to the hospital the temperature was 100. The blood count showed R. B. C. 4,640,000, hemoglobin 87 per cent, W. B. C. 11,050, polynuclears 73 segmented, 5 staffs. Roentgen examination of the nasal accessory sinuses was negative. Films taken of the mastoid processes showed no further disease of the resected areas. Both petrous pyramids were pneumatic and asymmetrical. There was, however, a marked decalcification of the petrous apex on the right side, with a distinct loss of the trabeculations. The contour in the region of the inferior petrosal sinus was not distinctly visualized.

On April 11, 1934, the child was again reoperated upon. The previous mastoid wound was reopened and the detritus in the mastoid cavity cleaned out. A search was made for a fistulous opening but none was found. The field was converted into a radical cavity and a fistula was found at the mouth of the eustachian tube. The granulations from the middle ear were removed and the fistulous opening was injected with lipiodol.

An x-ray of the petrosa, taken immediately after operation, showed the lipiodol to be distributed throughout the middle cranial fossa and the posterior clinoidal fossa above the tentorium on the right side, and also in the posterior fossa subdurally and into the spinal canal; and, further, it showed a perforation of the petrous apex. The site of this perforation was probably in the inner wall of the petrous pyramid either near or above the inferior pretrosal sinus.

The day following operation the temperature rose to 103 and from then it gradually fell so as to reach normal on the 19th of April. The pain in the eye had completely disappeared by April 14th. A blood transfusion of 100 cc. whole blood was given on the 14th. On April 23rd a plastic operation was performed on the right side and the left wound was recuretted and closed. A slight postoperative rise in temperature to 101.8 followed. The child was discharged from the hospital on April 26th.

The patient did well until May 20, 1934, when he complained of a sore throat, followed by some headache and a temperature of 100. These symptoms continued and the child was readmitted to the hospital on May 24th. On the date of admission the mother noted that the child appeared drowsy and complained of photophobia. Examination showed a slight nuchal rigidity. The blood count showed W. B. C. 18,400, polynuclears 76 segmented, 14 staff cells. Spinal fluid was cloudy, albumin and globulin 2 plus, copper reduction absent, 12,000 cells mostly polynuclears. Culture of the fluid yielded streptococcus hemolyticus pyogenes. A diagnosis of meningitis secondary to brain abscess was made.

The child was taken to the operating room and the right mastoid wound was reopened. There was some pus present in the tympanic portion of the radical

cavity but the remainder had epidermatized. The fistula into the petrosa was reentered with a probe but no pus was found. The entire tegmen was removed, exposing a markedly bulging dura. The dura was separated from the superior surface of the petrosa as far as the apex, but no extradural collection of pus was found. A large portion of the squama was next removed and an incision made into the dura in this area. A brain cannula was introduced into the temporosphenoidal lobe and at a depth of 4 cm. a capsule was encountered. This was quite thick as it required some force to penetrate it. When this was accomplished a gush of two ounces of creamy yellow pus followed. The abscess cavity was completely evacuated. The pus on culture yielded streptococcus hemolyticus.

The child was transfused with 250 cc. whole blood on the same day, and again on the 26th (115 cc.), the 28th (100 cc.), the 30th (100 cc.), June 1st, 3rd, 5th (each time 100 cc.).

On May 26th a partial paresis of the right external rectus appeared. General frontal headache continued. Bilateral Kernig present.

On May 28th the left external rectus became paralyzed. Despite repeated therapeutic spinal taps, intravenous administration of hypotonic solutions for the promotion of increased flow of spinal fluid, the child remained irrational and the spinal fluid continued to show the picture given above. The temperature, which was between 102 and 103 from the time of admission to June 30th, then fell to between 99 and 101. The child died on June 5th.

Comment. This case, when operated upon by us, already presented a perforation of the bony confines of the petrosa, as was demonstrated in the roentgenogram. The child recovered from its lesion in the petrosal pyramid. The ear became dry; the wound healed. Later there developed marked signs of intracranial pressure which were caused by a cerebral abscess. This abscess had a very thick capsule and was undoubtedly of long duration. In the absence of an autopsy further comment upon the case is impossible. We report it as one of the deaths from a petrosal lesion, but among our staff are some who consider the petrosal lesion recovered from and death due to a brain abscess which, like the petrosal lesion, was secondary to the otitic infection. This, however, is a debatable point.

CASE 7.—A. M., male, 10 years old, contracted a very malignant form of scarlet fever in the latter part of March, 1933. On the second day of the illness he developed a streptococcus cellulitis and cervical adenitis which later broke down and was incised. Then, during the first week of his illness, he also developed a sphenoidal and ethmoidal infection with severe edema over the right side of the face. Later he developed an edema from his forehead down to his third rib with very difficult breathing, and he was very toxic. He received 180 cc. of scarlet fever antitoxin in the first two days and two transfusions of 400 cc. convalescent blood. His left ear started to discharge spontaneously on the fifth day of the disease. The temperature ranged between 100 and 106, and three days after the onset of the otorrhea a left simple mastoidectomy was performed by Dr. Lederer of Chicago.

A very stormy postoperative course ensued. With the aid of transfusions and arsenicals, the temperature eventually returned to normal and the marked secondary anemia improved. For the next four months both the middle ear and the mastoid

Fig. 3. Case 7.—Base plate showing lipiodol in petrous apex.

wound continued to present a thick green mucopus, and the child complained of headaches on the right side, malaise weakness in the extremities, nausea and vomiting. A secondary mastoid operation was undertaken by Dr. Lederer on July 6, 1933; the dura and lateral sinus were exposed and appeared normal. This procedure did not suffice, for since that time the child became worse. On admission to Beth Israel Hospital the child complained of weakness, profuse discharge from middle ear and mastoid, nausea, vomiting, loss of weight, anorexia and pain in the left eye region.

The patient was admitted to the hospital on August 30, 1933. Temperature was 99.8. The patient was observed for the next two days. Roentgenogram of the temporal bone showed the apex of the left petrous pyramid to be partially decalcified—probably due to an inflammatory osteoporosis. The blood count was within normal limits.

On September 2, 1933, a preoperative diagnosis of empyema of the left petrous apex having been made, the patient was again operated upon by us. The old mastoid wound was reopened. The previous operation had exposed the lateral sinus and dura. A search was made for a fistula in the region of the epitympanic space, antrum and peribulbar area but none was found. A radical mastoidectomy was then performed. A thorough toilet of the middle ear was performed. It was then found that there was a fistula at the tubotympanic orifice leading into the petrosal tip. A probe introduced into this fistula encountered resistance which, when broken through, was followed by a spurt of pus. This welled up to fill the middle ear cavity. The petrosal tip was then curetted with a Yankauer curette and washed. Lipiodol was injected into the cavity.

The postoperative course was uneventful. All symptoms immediately disappeared and the child began to gain rapidly. The temperature stayed within normal range. On September 15th a flap was cut in the posterior membranous canal wall and the posterior wound closed. The child, at this writing, is completely recovered.

Comment. The finding of a fistula at the orifice of the eustachian tube and its proper management afforded adequate drainage and sufficed for a cure. It would not have been discovered by any technic which would have avoided the performance of a radical mastoidectomy. In the excellent monograph of Ramadier,[4] he describes in detail the various locations within the perilabyrinthine areas where suppuration may be found. In all, he lists six such sites: Underneath the semicircular canals, behind the semicircular canals, between the semicirculars, underneath the cochlea, anterior to and underneath the cochlea, and the apex. Consequently, the advocacy of *any one procedure* which will successfully attack *all these areas* is impossible. Therein lies a great source of misunderstanding. Ramadier, who described the technic for apical lesions, performed his own operation in four cases, while in his others he obtained a cure through the adequate handling of the fistulæ without resort to other surgery, exactly as we do.

The otologist who operates upon a definite case of petrous suppuration must reason out an intelligent plan of approach. Through

Fig. 4. Case 7.—Stenvers position. Lipiodol in apex.

the simple mastoid exposure he may be able to locate disease around the semicircular canals, exenterate diseased perilabyrinthine cells and discover fistulous openings in the peribulbar area beneath the posterior semicircular, behind the superior semicircular and through a wider exposure of the aditus, in front of the superior semicircular. Failing to find any openings in these areas, or in the event that the management of these, when found, does not effect a cure, it is imperative that the tympanic spaces be opened and the inner wall of the middle ear carefully inspected for fistulæ below the promontory and also in the region of the carotid canal, particularly at the mouth of the eustachian tube. Failing here, a direct attack of the apex can be made by the methods advocated by Eagleton, Ramadier or Almour. In a previous paper, read by one of us (Kopetzky),[5] the merits of the various classical procedures were compared. Our advocacy of the Almour technic continues to find substantiation as the one most fitted for the closed empyemas of the apex. Our results speak for themselves.

Case 8.—S. S., male, age 8 years, was admitted to the Beth Israel Hospital on May 22, 1933. Two and one-half weeks prior to this he complained of pain in his left ear; the drum ruptured spontaneously and there has been a profuse otorrhea since. Just prior to admission the child had a chill and a temperature of 104.

Otoscopic examination revealed a profuse purulent discharge from the left middle ear, which welled up under jugular compression. There was some tip tenderness and the child appeared lethargic. Blood count on admission was R. B. C. 4,120,000, hemoglobin 73 per cent, W. B. C. 12,100, polynuclears 72 segmented, 2 staffs, mononuclears 25. Roentgen examination on May 23rd revealed a destructive process in the left mastoid; on the left side the apex of the petrous pyramid showed a slight degree of osteoporosis but no destructive bone changes.

On May 26th, after several days in which the temperature fluctuated between 102 and 104.2, a left simple mastoidectomy was performed. The mastoid was partly diploic and partly pneumatic. The sinus was relatively far forward. The antral cells were filled with greenish looking granulations. The tegmen near the zygomatic root was covered by a layer of diploic spaces filled with thick white pus which yielded streptococcus hemolyticus equi on culture. The sinus was uncovered in view of the septic temperature but appeared normal.

During the following nine days the temperature dropped to a lower level but still reached 102 plus once daily. The day after operation there was a marked rise in the W. B. C. to 34,000, 89 polynuclears, of which 10 were staff cells. This gradually dropped on the succeeding days.

On June 10th, after the temperature had reached the 100 level, the child complained of severe pain in the left eye, lasting 15 minutes. Up to this time the dressings revealed a satisfactory healing of the mastoid wound but a continuing profuse otorrhea. A slight paresis of the left external rectus was noted on June 15th. A roentgenogram of the petrosa, taken on June 17th, showed further decalcification of the left petrous pyramid since the examination on May 23rd.

On June 20th, the left mastoid wound was reopened and the cavity found in good condition. The wound was converted into a radical cavity and the middle ear

Fig. 5. Case 8.—Stenvers position. Lipiodol in petrous apex.

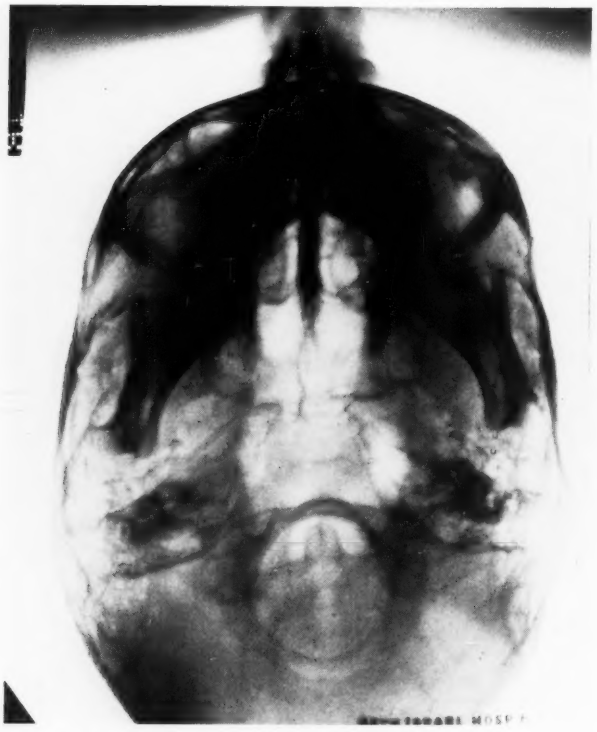
found full of granulations. A search for a fistula was made, but none found. The anterior buttress was removed and the tensor tympani muscle ablated. The petrous apex was entered, according to the Almour technic, and a gush of pus followed by some blood ensued. Some diseased bone was found in the solid angle and was removed. An impairment of function in the left facial was noted upon coming out of anesthesia.

On the 22nd of June, the left abducens palsy was complete. Some eye pain was still complained of. The rest of the postoperative course was uneventful. The child was kept in the hospital until July 28th, during which time the abducens palsy completely cleared and the facial paralysis improved under physical therapy. The temperature during the entire time was within normal limits. At the present writing, the radical cavity is completely healed and the lower two branches of the facial have recovered their function completely, while the upper branch is still affected to a slight degree.

Comment. Here was a case of closed empyema of the apex. The method of attack outlined in the comment on the preceding case was followed. No opening into the petrosa was found. One was then made, using the Almour technic; pus was obtained and a cure effected. The facial nerve was probably traumatized during the curettage in the vicinity of the so-called solid angle. The abducens palsy present in this case must be considered as the direct result of the suppurative lesion within the apex.

CASE 9.—L. S., male, 28 years old, was referred to us by Dr. G. G. Hankins of Newport News, Va., with the following letter: "There was a past history of right chronic mastoiditis for about two years. This past summer has drained a great deal and about September 1, 1933, patient complained of pain in right side of head about the temporal region and a radical mastoidectomy was advised. On September 26, 1933, this operation was performed and the mastoid wound healed in due time without any special difficulty. While convalescing in the hospital patient continued to complain of pain in the right temporal region and ran a low temperature at frequent intervals. He returned to his home and the ear was dressed at the office for a week or more when the patient complained of severe pain in the right temporal region and back of the right eye, and could not sleep without codein and ice bag. October 16, 1933: White count 14,000, polys 76, small lymphs 16, large lymphs 6, eosinophiles 2. October 17, 1933, patient consulted Dr. Dandy at Johns Hopkins, where he was observed for about one week, and returned with the opinion that he would have no further trouble. About November 3rd patient complained of some pain in the right temporal region and right eye, and the ear discharged thick mucoid pus more abundantly than for several days. Since that time the pain has been more or less constant and radiating to the back of neck. Temperature has ranged from 99.5 to 101. The discharge from the ear can be traced to the region of the eustachian orifice. X-ray plates show some haziness in the petrous pyramid. Diagnosis: Petrositis."

The patient was seen by us on November 14th and sent to Beth Israel Hospital for observation and study. Roentgenogram reported by Dr. H. K. Taylor showed an infection in the right petrous pyramid with a suppurative lesion in the petrous apex. There was no apparent perforation of the petrous apex.



Right Left

Fig. 6. Case 9.—Rarefaction and destruction of cell septa on right side.

For the following three days the temperature ranged between 100 and 101; and the pain in and about the eye and in the right temple increased in severity. Neurologic and ophthalmologic examinations revealed normal findings. The blood count and urine were normal. On November 15th, after cleaning the left radical cavity and thoroughly drying it, a probe pushed inward toward the eustachian orifice engaged itself into a cavity; this was followed by a free flow of pus which was thick and creamy in appearance. The patient at this time told us that he was a sufferer of bronchial asthma.

On November 17th, the radical mastoidectomy was revised and the middle ear cleaned out. A fistula was found at the mouth of the eustachian tube at the exact point where we make our entrance of selection into the petrous tip. This fistula was enlarged with a burr and lipiodol was injected into it. The wound was left open.

The patient continued to have pain, decreasing in severity, over the forehead and temple on the right side, until November 21st. On November 24th, the wound was closed and, beside the slight discomfort of wound and neck pain, the man felt well. He was discharged from the hospital on November 28th. He has fully recovered, the ear has dried and all symptoms have disappeared.

Comment. Here again, as in Case 3, a fistulous opening was found during a dressing. The fistula, located at the orifice of the eustachian tube, was not sufficient for adequate drainage. Only after enlargement and curettage of the apex was a cure obtained.

CASE 10.—F. C., female, 10 years old, fell ill with a cold in the early part of December, 1933. On the 10th of that month she experienced pain in the right ear and a myringotomy was performed two days later. The ear discharged profusely until the day of admission to Beth Israel Hospital, December 21, 1933.

The temperature on admission was 102.4. The blood count showed R. B. C. 4,000,000, hemoglobin 84 per cent, W. B. C. 13,300, polynuclears 74 segmented, 3 staff cells. Otoscopic examination showed the classical picture, in the fundus, of an acute coalescent mastoiditis. There was an exhaustible ankle clonus on the left. There was a suggestive Kernig on the right side. The child complained of generalized headache. The roentgen films taken on the date of admission showed a coalescent lesion in the right mastoid process. The aeration of the cells in the right petrous tip was markedly diminished, but there was no evidence of bone destruction in the petrosal portion of the temporal bone.

On December 22nd, a right simple mastoidectomy was performed. A coalescent mastoiditis in an extensively pneumatized bone was found. There was an unusual extension of the infection into the zygomatic roots. A large development of cellular structure, which was infected, was located behind the semicircular canals. A complete exenteration of the diseased tissue was performed and the inner table exposed throughout; the antrum was opened. Culture of pus showed streptococcus hemolyticus pyogenes.

After a postoperative rise to 103.2, the temperature for the following week ranged between 100 and 101.4; on December 28th to 29th, 99 to 101.8; on the 30th it rose to 102.6 and then returned to its former level, and on the date of discharge from the hospital was 100.2. There were no untoward symptoms during the postoperative period. The middle ear and mastoid process continued to discharge pro-

Fig. 7. Case 9.—Postoperative injection of lipiodol through fistula with collection of chemical in petrous apex.

fusely for the week following operation but then gradually lessened in amount. On December 31st, a return of the left ankle clonus was noted, but this disappeared the following day.

The child was dressed every other day from the date of discharge until March 28, 1934, a period of 83 days. During this time the middle ear dried and remained so. The mastoid wound, however, presented a constant thick, purulent discharge from the lower angle, with exuberant granulations repeatedly forming despite numerous curettings and caustic applications. The child, during this period, experienced no pain whatsoever nor did she exhibit any signs of labyrinthine irritation. She did, however, on several occasions, have a rise in temperature, sometimes as high as 101.4. Even then the child was bright and she did not lose weight.

We then felt that we were dealing with a lesion which, in the presence of a pneumatized temporal bone, was responsible for a chronic mastoid lesion. An overlooked or a continuing necrosis of the inner table or, more probably, the presence of a suppurative process in the petrous pyramid was responsible for the chronicity.

A roentgenogram of the mastoid process on March 26, 1934, ruled out further disease in that cavity. The right petrosa, however, showed a marked diminution in aeration; the trabeculæ in the apical portion were visualized and the apical contour was intact. There was no atrophy of the petrous apex. The diagnosis of Dr. H. K. Taylor was "suppurative petrositis with adequate drainage."

The child was readmitted to the hospital on March 29, 1934. Temperature on admission was 99.8. Blood count showed normal findings. The child was re-operated on tne date of admission. The mastoid wound was reopened and sickly granulations were removed from the wound. A piece of necrotic tegmen was found and removed. A wide exposure of the aditus was made, and a search for a fistula revealed no opening into the petrosa in the areas behind and in front of the superior semicircular canal. Search was then made in the peribulbar area beneath the posterior semicircular canal and a fistulous tract was found leading into the petrosa. A probe inserted into the tract took a direction inward, upward and forward. Lipiodol was injected and during this procedure one and one-half teaspoonsful of thick yellow pus escaped which yielded a streptococcus on culture. The fistula was widened and drained with rubber dam.

For the next four days the child complained of pain in the teeth on the right side and also of slight supra- and intra-orbital pain. This had completely disappeared on the date of discharge, April 2, 1934. Roentgenogram immediately after operation showed a large puddle of lipiodol in the region corresponding to the right petrous apex.

From then on, the healing of the mastoid wound was rapid. The child made an uneventful recovery.

Comment. This is a definite case of chronic suppurative petrositis. None of the classical signs or symptoms, with the possible exception of a low grade sepsis, was present at any time. The adequate drainage afforded by the fistulous opening in the peribulbar area was sufficient to safeguard the child during this time from the danger of intracranial involvement. It likewise prevented pressure of pent-

up pus in the petrosa and so accounted for the absence of eye pain. That this is true can be shown by the appearance of trigeminal irritation after the forcible injection of lipiodol into the petrosa.

There is much discussion at present as to whether some cases of suppurative petrositis recover without resort to surgery. We will grant that this child's life was safe, even in the presence of an active suppuration in the petrous apex. Nevertheless, with a teaspoonful and a half of pus within the petrosa, we doubt very much if this child would ever have had her mastoid wound healed. She would have gone on with a chronic lesion until a blockage by granulations would have eventually brought on the symptoms of a petrosal lesion.

As far as the performance of surgery in all cases of petrous suppuration is concerned, we take the following stand: Every case of acute and subacute petrous suppuration should be drained. Every case of chronic suppuration of the petrosa should also be drained in order to clear up the chronicity and obviate continuing osteitis.

A radical mastoidectomy was not required in this case because the middle ear had entirely healed, thus indicating the presence of a fistulous tract within the area accessible by a simple mastoidectomy.

CONCLUSIONS.

Summarizing, we have heretofore reported seventeen cases. Herewith we report ten additional cases, of which nine presented fistulous tracts leading into the petrosal apex.

In four cases simple mastoidectomy with adequate widening of the fistula and drainage sufficed for a cure. In six cases radical mastoidectomy was necessary to reach the site of the fistulous opening.

In two cases a sixth nerve palsy was present prior to exploration of the petrous apex.

In one case a sixth nerve palsy appeared subsequent to drainage of the petrous apex.

In two cases there was proven endocranial rupture of the lesion in the petrosal apex by lipiodol injection.

In one case a lateral pharyngeal abscess of petrosal origin appeared simultaneous with the appearance of a sixth nerve palsy.

Of the cases here reported, one died and nine recovered. Five other cases, seen and operated on by us but not reported here, recovered.

RECAPITULATION.

In all we have had thirty-two proven cases of suppuration within the petrosal pyramid. Twenty-seven of these have been cured by the surgical attack of the lesion within the petrous apex.

One death occurred in a patient who was not operated upon and in which case the lesion was not recognized (the first case studied by us).

Of the other four deaths, three patients were operated upon by us in the presence of a fully developed purulent leptomeningitis. The remaining patient succumbed to a brain abscess.

All these cases were proven by operative findings, by lipiodol injections and by roentgenograms taken postoperatively, showing the lipiodol in the petrosa. (In the first patient seen by us, who died, the condition was proven by autopsy.)

51 WEST 73RD STREET.

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VI.

SOME CONCLUSIVE REMARKS REGARDING PLASTIC SURGERY FROM PERSONAL EXPERIENCE.*

JOSEPH C. BECK, M. D.,

CHICAGO.

A recent visit to this country by Sir Harold D. Gillies, of London, brought him to Chicago, where he delivered the annual Charles Mayo Northwestern University lecture.

He chose the subject of "Plastic Surgery," and the writer had the pleasure of hearing it. It was what he said regarding the possible future of plastic surgery and the duty of the surgeon to the public that prompts me to bring this subject before you for your consideration. Gillies is considered one of the outstanding contemporaries in this work, and having watched it from the time I visited him and his staff in 1918, during the World War, at Queen's Hospital in Sidcup, England, and again in London in 1927, as well as having read his book on Plastic Surgery, which is a classic, I feel sure that whatever he has to say about the subject can be relied upon. Therefore, when he made the pertinent statement that the majority of surgeons have the wrong slant on the indication for cosmetic surgery, I was compelled to take notice. I do not believe it is necessary for me to go into any detail to establish my interest and familiarity with the subject of plastic surgery, as I have done considerable work along this line, as far back as 1894, when the great Nicholas Senn was doing reconstructive plastic surgery and I had the pleasure of dressing some of his cases.

In 1908, I presented a series of cases of plastic surgery before the American Laryngological, Rhinological and Otological Society. Most of them were of the major reconstruction variety, although there were a few cosmetic cases among them. In my paper I asked for a frank discussion as to whether the members thought that this type of surgery was proper for our specialty, because I had heard that some men considered it questionable or bordering upon the unethical. One will note that the writer at that time expressed himself similarly to Dr. Gillies' recent utterances.

*Presented before the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society at Indianapolis, January 23, 1935.

Permit me to quote from the transactions of 1908 of that Society: "This subject has been and is yet, to a great extent, treated by the general surgeon, notwithstanding the fact that detailed intranasal surgery is often necessary to obtain the best results. Again, a large majority of external nasal deformities are treated by charlatans because general surgeons, as well as rhinologists, often refuse and discourage treatment of the above named conditions for cosmetic purposes. This fact, I believe, is responsible for many bad results, accidents and malformations, rather than improvements. Many such patients belong in the care of a neurologist, and should receive the strictest attention, and by all means prevent them from falling into the hands of an unskilled so-called specialist and beauty doctor.

"Those of you who have seen some of these cases after they were corrected by these so-called specialists wonder if you have not acted unwisely by refusing to listen to these unfortunate individuals."

Since 1908 I have refused to do cosmetic borderline operations in many instances, thus falling under that group of surgeons that Gillies criticises. However, I have done a fair number of reductions and building up of parts about the head and neck that are decidedly cosmetic.

Today, instead of demonstrating technical points of operations, I thought it would be more interesting and less time consuming if I presented very briefly some of my cases that are waiting for work to be done at the present time, and bring out for discussion some of the difficulties encountered. One will notice that several of these patients are wearing a prothesis because their conditions are inoperable. This phase of the subject deserves our great consideration. For obvious reasons most of these patients cannot be presented in person. Therefore permit me to outline the problems involved. (Here a moving picture was shown.)

Case 1. Mrs. D.—Frontal sinus deformity, postosteomyelitic.

Case 2. Miss B.—Complete unilateral soft palate defect secondary to scarlet fever and measles as a child.

Case 3. Miss M.—Bilateral malformation of nostrils and imperfect repair of harelip—complete wide cleft palate.

Case 4. Baby R.—Minute central fistula in a perfect repair of harelip and cleft palate with lateral nasal deformity.

Case 5. Mrs. K.—Minute retroauricular fistula secondary to osteomyelitis of mastoid with plastic repair.

Case 6. Miss D.—Saddle nose with expulsion of ivory at two different times.

Case 7. Miss W.—Laryngostomy for postscarlatina atresia of both vocal cords as an infant, now showing the complete technic of closure in three stages.

Case 8. Mr. S.—Esophageal fistula; postlaryngectomy.

Case 9. Miss N.—Lips and chin wrinkling in a young individual following application of an ointment.

Case 10. Master R.—Large skull defect secondary to operation for osteomyelitis of the frontal sinus and calvarium.

Case 11. Mrs. J.—Facial paralysis and ankylosis of lower jaw following osteomyelitis of the temporal bone.

Case 12. Master B.—Ectropion of the lower lid secondary to osteomyelitis of the orbital margin from maxillary sinus disease.

Case 13. Miss D.—Saddle nose; trauma following automobile accident.

Case 14. Mr. T.—Saddle nose and multiple fistula with granulations; intranasal and sinus tuberculosis.

Case 15. Mr. F.—Loss of both eyes, complete loss of the external and intranasal structures and part of upper lip due to posttraumatic elevator accident.

Today I am convinced that cosmetic surgery has come to stay and not only should we be willing to do the work but rather encourage it. There are so many unscrupulous men in this field that the earlier we let the public know we are doing this work the earlier will the poorly prepared advertising quack be driven out. When I say we should let the public know that we do this type of work I do not mean by press advertising or through any kind of lay literature.

One very important point is to make it possible for the average individual to have this work done. Thus far, like prosthetic dentistry, only the well-to-do can afford to have plastic operations performed. I believe that this is wrong and can be corrected. Many of these individuals will sell or borrow to get money enough to have this work done, especially that most difficult individual, the psychoneurotic. Here is where one should make a sharp distinction as to whether to operate or not, because it has been my experience, as well as that of others, that no matter how good a result is obtained most of these individuals are never satisfied. However, I have seen cases where the work was so unsatisfactory that it is not at all surprising that the patients are dissatisfied. This occurs especially when the work is done by an occasional operator in plastic surgery.

While I hope that this work may become a regular rhinologic procedure and more widely spread, I believe plastic operations should be performed only by those who have prepared themselves in this field. Therefore, I would urge the establishment of clinics devoted

especially to plastic surgery and only in our regular teaching institutions.

The technical side of plastic surgery is, of course, most important and new ideas are constantly presented. The literature, especially in the English language, is so voluminous that any reference on my part about the technic would surely be out of place.

I am confident that anyone interested in plastic surgery can obtain advice, instruction or even courses in plastic reconstruction or cosmetic surgery.

To be sure, such courses are not easily obtained at this time because most of the patients are seen in private practice and are somewhat supersensitive when demonstrated. Therefore, I repeat that there is a need of plastic surgery clinics in reconstructive as well as cosmetic operations within our teaching institutions.

Maliniak has just published a book entitled "Sculpture in the Living," which I have perused and found very interesting and instructive. The title appears quite "catchy," but since it is for the general practitioner and not the layman, I am sure it can do only good.

What about the irregular plastic surgeon? Throughout this country there are men and women who place enticing advertising in the lay press magazines, in beauty shops and newspapers, and use many other less dignified yet nevertheless clever methods of attracting clients.

Two years ago a plastic surgeon told me that there were thirty-two individuals doing plastic operations in and about one city, and only two of these were members of the regular local medical societies. It is well for the officers of local medical societies to be on the lookout for some of these individuals who are attempting to obtain membership in the various societies for advertising purposes.

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VII.

EXPERIENCES IN IONIZATION OF THE NASAL MUCOUS MEMBRANE.*

HAROLD G. TOBEY, M. D.,

BOSTON.

The theory of ionization of mucous membranes by metallic solutions aided by an electrical current has been applied for more than a decade, but has not been widely practiced. A new impetus has been given to this therapeutic measure through the work of H. L. Warwick, who has investigated the results to be obtained from various metallic solutions and their combinations, and has developed a means by which a definitely controlled smooth direct current of electricity may be passed through the structures to be treated without harm to the tissues or discomfort to the subject.

The theory, briefly stated, is that a metallic solution placed in contact with the nasal mucous membrane results in the precipitation of the proteins of the superficial cells, but when a mild direct current is passed through the tissues the metallic ions penetrate more deeply and precipitate more of the proteins of the cells. Great care is taken that the strength of the current does not reach the point of coagulation of the tissues.

Warwick, after considerable experimentation, has adopted a solution consisting of sulphate of zinc 85 per cent, tin 10 per cent and cadmium 5 per cent, as best suited for the treatment. An alloy of these three metals, in the same proportions, is used for the anode.

The method of treatment consists in thoroughly anesthetizing the entire mucous membrane, following which, the nasal cavities are thoroughly packed with cotton strip saturated with the metallic solution and the anode introduced. The cathode is then affixed to the arm of the same side. The efficiency of the packing is indicated by a resistance ohmeter which is incorporated in the instrument. The resistance should be from 2500 to 3000 for a satisfactory result. If this resistance should be as high as 8,000 to 10,000 ohms it is positive evidence that the nasal packing is inadequate.

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*Presented before the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Portland, Me., January 4, 1935.

The direct current is then turned on slowly to the desired strength. One hundred (100) milliampere-minutes—that is, ten milliamperes for ten minutes—has been found to give good results. However, the strength of the current may be varied according to circumstances, and as dictated by experience.

The immediate discomfort to the subject is negligible and consists of having the nasal cavities tightly packed and a metallic taste as the current reaches its maximum. The after results are more troublesome. Soon after the completion of the treatment there ensues a period varying, usually, from eight to twelve hours, when there is an intense burning sensation in the nose, eyes and face accompanied by some pain and headache. In addition there is frequently a marked nervous reaction, the extent of which is dependent on the temperament of the subject.

The treatment has been developed primarily for the alleviation of hay fever and seems to be particularly applicable to those vasomotor disturbances of the nasal mucous membranes which are today usually classed as allergic.

Our experience, so far, has consisted of the treatment of sixteen subjects with a conservative general estimate of 85.6 per cent of relief.

Eight of these suffered from hay fever and in this group there was 85.6 per cent of relief. One subject suffering from fall hay fever was treated in June with the result that he was entirely free for the first half of the season, after which the usual symptoms intervened with their usual severity. All patients treated after the onset of the symptoms were free of all discomfort thereafter.

Eight were the victims of an hyperesthetic rhinitis and, among these, three were also sufferers from asthma. Two of the latter had already been relieved of asthma before the treatment was instituted, while in the third there was no relief following the treatment. In this group it will be noticed that while there was 83.7 per cent of immediate relief, the time element begins to make itself manifest.

Case 10 suffered from asthma, urticaria and hyperesthetic rhinitis for two years when first seen two years ago. She has been relieved of asthma now for one and a half years, and of the urticaria for one year. All efforts at relieving the rhinitis had proved of no avail up to May, 1934, when the Warwick treatment gave complete relief for six months. The second treatment has just been completed so that

the result is not yet available. The causative agent seems to be heavy perfume, which she has no way of avoiding since her livelihood depends upon the teaching of music.

Case 11 experienced about 50 per cent of relief for a period of two months.

Case 9 has had 50 per cent relief for the full six months following the treatment.

The other cases have experienced immediate relief, but a sufficient time has not as yet elapsed for any final decision.

The change in the appearance of the mucous membrane is as striking as the relief from symptoms. Following the stage of primary congestion, the mucous membrane takes on the color of normal healthy lining, which is in great contrast to the pale, boggy appearance before treatment.

Case	Age	Dates of Ionization	For What Was Ionization H. F.	H. H.	Asth.	Duration of Symptoms	Result of Ionization	Remarks
1	23	June '34	*	....	....	2 yrs.	50%	Fall hay fever treated in June
2	60	Sept. '34	*	....	....	40 yrs.	100%	.....
3	40	Aug. '34	*	....	....	yrs.	100%	Relief Total 16 85.6%
4	55	Aug. '34	*	....	....	yrs.	85%	Hay F. 8 87.5%
5	7	Oct. '34	*	....	....	5 yrs.	75%	Hyp. R. 8 83.7%
6	12	Oct. '34	*	....	....	3 yrs.	100%	.....
7	55	Sept. '34	*	....	....	yrs.	100%	.....
8	16	Aug. '34	*	....	....	10 yrs.	90%	.....
9	50	May '34	....	*	....	10 yrs.	50%	.....
10	48	May '34	....	*	*	4 yrs.	100%	Arthma, urticaria H. R. relieved 6 mos.
11	43	Aug. '34	....	*	....	3 yrs.	50%	Two months
12	45	Oct. '34	....	*	*	17 yrs.	100%	No relief to asthma
13	20	Aug. '34	....	*	....	3 yrs.	75%	.....
14	18	June '34	....	*	....	3 mos.	100%	.....
15	52	Aug. '34	....	*	*	10 yrs.	95%	Asthma, Migraine, Urticaria
16	40	Nov. '34	....	*	....	3 yrs.	100%	.....
			8	8	3			

CONCLUSIONS.

1. The method is efficient for the control of hay fever if instituted after the onset of the symptoms. We feel, however, that desensitization should be the method of choice.

2. The immediate results in hyperesthetic or vasomotor rhinitis are excellent. The duration of these results is as yet unknown.

270 COMMONWEALTH AVE.

VIII.

DIAGNOSIS OF ACUTE SUPPURATION OF THE PETROUS PYRAMID.*

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Suppuration of the petrous pyramid is the most recently recognized complication of purulent otitis media. It is caused by extension of the infection from the middle ear or mastoid by way of the cell channels leading into the petrous pyramid.

Previous to 1929, very few cases were diagnosed before autopsy. During the period from 1929 to 1931, Kopetzky and Almour,[1] Eagleton,[2] Profant,[3] Friesner and Druss[4] and Voss[5] were among the first to formulate a clinical picture of this condition. Ramadier[6] has published the most extensive treatise appearing since 1931. Credit for the stimulation of this work is due to those men who studied the causes of sixth nerve paralysis. Among those who contributed much may be mentioned Dorello[7] in 1906, Wheeler[8] in 1918, Vail[9] in 1923 and Sears[10] in 1925.

At autopsy on patients dying from otitic meningitis, careful scrutiny frequently revealed necrotic areas on the surface of the petrous pyramid, thus emphasizing the urgent need for diagnosis of the pre-existing suppuration.

The many discussions in the recent literature establish the fact that Gradenigo's syndrome, consisting of paralysis of the external rectus muscle and unilateral temporoparietal pains in the presence of acute otitis media of the affected side does not typify a clinical entity, and should not be confused with the picture presented by petrous suppuration. It is the belief of Kopetzky,[11] Eagleton[12] and a large number of other prominent otologists that often Gradenigo's triad of symptoms is the result of extension of the infection by way of the veins—retrograde thrombophlebitis or is toxic in origin, factors entirely outside the petrous pyramid, causing a mild localized area of meningitis involving the dura in contact with the Gasserian

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ganglion and sixth nerve. Supporting this opinion is the fact that unilateral temporoparietal pain and sixth nerve paralysis appear as early symptoms, sometimes before suppuration in the mastoid is established, and in rapid sequence, whereas, if abducent paralysis does occur with empyema of the petrous pyramid, it is usually a later symptom, appearing when the infection has reached the tip cells of the pyramid.

ANATOMY.

The following brief consideration of the anatomic relations associated with the symptomatology will help clarify its interpretation.

Bone.—The acute cases of suppuration of the petrous pyramid occur most frequently in pneumatized mastoid bones. Extensively pneumatized temporal bones present cellular development extending into the zygoma, the squamo, the occipital bone, the floor of the middle ear, around the mouth of the eustachian canal, well down into the anterior wall of the mastoid tip and into the petrosal pyramid. (Figs. 1, 2, 3, 4, 5.)

Carmack,[13] in a dissection study of ninety-four petrous pyramids in adults, found 32 per cent pneumatized. He states, "There is every anatomic reason for extension of an inflammatory process into the cells of the pyramid, with otitis media, similar to that occurring in a mastoid process."

Hagens[14] studied fifty temporal bones and found 34 per cent had cell spaces in the petrous pyramid.

Myerson, Rubin and Gilbert[15] examined 200 temporal bones, both before and after removal from the cadavers and, by gross section, found only 11 per cent pneumatized petrous tips. This low percentage is largely due to the twenty-three specimens from infants. An interesting finding in their series is that fifty-four temporal bones from negroes showed three times the percentage of cellular petrous pyramids as did those in white subjects.

Glick[16] states that "pneumatic cells are present in the apex of the human temporal bone more frequently than is generally believed." In a microscopic study of the petrous apex, he found that inflammatory changes extended with less proliferative activity in the marrow than in the lining membrane of the cells.

Eagleton[17] has found infection extending by way of marrow-filled cancellous tissue into the petrous apex.

Fig. 2. Extensive pneumatization of temporal bone. From collection of E. B. Burchell.

Fig. 1. Temporal bone with pneumatization of zygoma and squama. From collection of E. B. Burchell.

Pathways.—The most generally recognized cellular pathways leading into the petrous pyramid are those described by Kopetzky.[18] (Fig. 6.)

1. From the anterior or tympanic space above or behind the superior semicircular canal, following the posterosuperior surface of the petrous pyramid into the tip.

2. From the peritubal cells into the pyramid.

3. From the peritubal cells by way of the carotid canal into the tip.

Ramadier,[27] in an extensive treatise in 1933, pictures the entrance of six avenues of infection as follows: (1) Prebulbar, (2) pretubal, (3) sublabyrinthine, (4) postlabyrinthine and (5) supralabyrinthine.

Nerves.—The great superficial petrosal nerve, which in all probability carries sensory as well as motor fibers, according to Piersol,[19] arises from the facial nerve in its canal, comes to the surface of the petrous pyramid through the hiatus fallopi, passes usually in a groove on the surface of the petrous pyramid outside of the dura, for a distance of 8 to 10 mm., before disappearing under the Gasserian ganglion to reach the foramen lacerum, where it joins the great deep petrosal nerve to form the Vidian nerve, thence through the Vidian canal in the sphenoid bone, the ascending orbital branch passing through the sphenomaxillary foramen to supply the periosteum of the orbit, the posterior ethmoid cells, etc.

Vail,[20] speaking of great superficial petrosal nerve neuralgias, says: "This neuralgia is of extreme diagnostic importance when we are dealing with a case of suppurative otitis, because one should look upon the complaint of pain around the eye, back of the eye, in the temporal region and back of the head as pathognomonic of irritation somewhere in the course of the great superficial petrosal nerve and that place can only be in the petrous apex."

The trigeminus or fifth cranial nerve reaches the surface of the petrous bone through an opening in the dura after leaving the lateral side of the pons. The sensory roots form the Gasserian ganglion which is enclosed in Meckel's space, a delamination of dura lying in a slight depression on the apex of the petrous bone; internally, the Gasserian ganglion comes in relation with the cavernous sinus and internal carotid artery. Of the three branches given off from the Gasserian ganglion, we are concerned only with the first or ophthalmic branch.

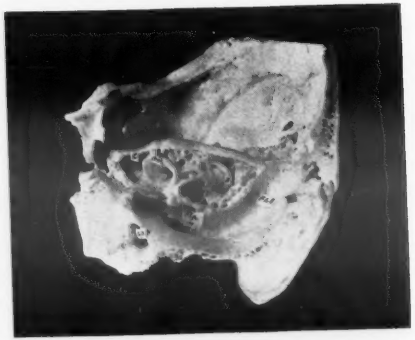

Fig. 3. Pneumatization of the petrous pyramid. From collection of E. B. Burchell.

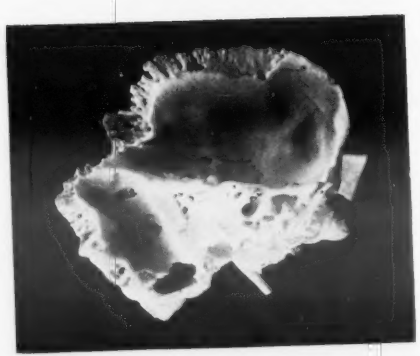

Fig. 4. Large cell in the apex of the petrous pyramid. From collection of E. B. Burchell.

This branch is more firmly bound down by the dura in its longer course within Meckel's space than either of the other two, subjecting it to greater tension when irritation of the Gasserian ganglion occurs; the ophthalmic nerve passes upward and forward in the external wall of the cavernous sinus for about 25 mm., thence through the sphenoid fissure to supply the lacrimal gland, the upper eyelid and the skin around the external canthus.

Abducent or Sixth Nerve.—This nerve is exclusively motor in function, supplying the external rectus muscle of the eye. Leaving the lower surface of the pons, it pierces the dura over the sphenoid bone, turns forward and passes between the apex of the petrous bone and posterior clinoid process of the sphenoid bone. A strong, fibrous bundle connecting these two bony prominences forms Dorello's canal. The superficial petrosal sinus as well as the abducent nerve passes through the canal. Passing forward and upward to the orbit, the abducens supplies the external rectus muscle.

SYMPTOMATOLOGY.

Aural Discharge.—The discharge from the ear is continuous from the onset of the otitis or reappears in from two to six weeks following mastoidectomy. In forty-eight cases collected from the literature, where the facts are noted it was continuous in thirty-three cases and reappeared in fifteen. In three cases of my own, two were acute with continuous discharge, and one, a chronic case, had intermittent discharge over a period of four years; was then quiescent for six years, until death from meningitis revealed, at autopsy, necrosis of the petrous apex. Many of these cases of continuous or reappearing discharge are reoperated upon one or more times, with the hope of locating an overlooked focus in the mastoid, only to discover eventually that the discharge is finding its way through a fistulous opening from the petrous bone into the middle ear or mastoid.

The bacteriology is not significant.

The continuous or reappearance of a profuse discharge, especially when accompanied by temperature and pains around the eye of the same side, two to six weeks after mastoidectomy, points strongly towards petrous pyramid suppuration.

Pain.—This is the most characteristic and constant symptom. It is located in, back of, and around the eye, in the temporoparietal region, sometimes reflected to the occiput of the affected side. At first it is usually nocturnal in character, described as a severe, agoniz-

Fig. 5. Pneumatized temporal bone with large cell in apex. From collection of E. B. Burchell.

ing, deep pain in the eye, with a feeling of the eye's being pressed forward. As the disease progresses, the periods of severe pain may change, occurring during the day, the severity of the pain lasting a few hours at a time. If questioned carefully, the patient is found rarely to be entirely free from pain in the parietal region.

Between the attacks of severe pain the patient is often fairly comfortable. If seen in the afternoon following a nocturnal attack it is hard to realize the extent of the suffering described by the patient, and he is put down as a neurotic. A note charted by the interne, of a patient whose condition later was diagnosed as petrous pyramid suppuration, reads: "She is about the worst neurotic I have ever seen." This nervous irritability is more or less characteristic of the disease.

This peculiar type of eye and associated pain persists and becomes more intensified until free drainage is afforded either by surgical means, by a large fistulous opening formed by necrosis into the middle ear or rupture of the cortical surface of the petrous apex. If the latter occurs, the patient goes into the quiescent stage and soon develops the terminal stage of meningitis.

Kopetzky[21] states of this peculiar eye pain: "It is highly significant of petrous pyramid suppuration," and explains it as the result of an irritation of the ophthalmic branch of the trigeminus where it is firmly bound down in its course from the Gasserian ganglion to and through the cavernous sinus.

Vail[22] writes: "The eye and orbital pain in cases of suppuration of the apex of the petrous bone is not due to the irritation of the Gasserian ganglion, but to the greater superficial petrosal nerve."

Eagleton,[23] discussing Vail's paper, is unable to say from his cases which nerve causes pain; probably both the fifth and the Vidian nerves are responsible in different cases, depending upon the position of the lesion and type of bone.

Owing to the position of the great superficial petrosal nerve in a groove in the petrosal cortex, outside the dura, and 10 mm. nearer the origin of the infection, it seems logical to believe that the earlier pains come from its irritation. As the infection extends further into the apex it reaches the cells underlying the Gasserian ganglion and irritation of the ophthalmic nerve takes place. It is not so important to recognize which of the two nerves carry the pain impulses as it is to be able to interpret the cause of the pains. In many pneumatized petrous bones the cells do not extend to the apex and the irritation

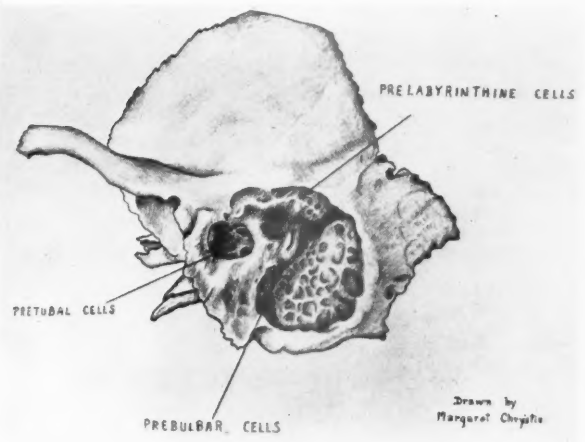

Fig. 6. Areas of entrance of cell pathways.

reflected through the thin cortex of the bone does not reach the Gasserian ganglion. If a localized leptomeningitis takes place over an infected petrous pyramid, as many authors believe, then both nerves are involved.

Temperature of the low grade, septic type rarely exceeds 102° F. and usually ranges from 99° to 100° F. during the day. There may be periods of several days with nearly normal temperature. The temperature remains of the above type until the terminal period of meningitis or until the suppuration is relieved by drainage.

The *blood picture* is of little value except to show that a mild infection is present, causing an increase of leucocytes from 10,000 to 15,000, the picture of a subacute infection.

The eye grounds usually show no change.

Roentgen Ray.—Too much dependence should not be placed upon the roentgen ray findings. One of my recent cases of operative mastoiditis of the right side showed increased density of the left petrous bone at the first exposure.

Coates, Ersner and Myers[25] report nine cases of roentgenographic changes in the petrous bone, some of them without clinical manifestations. Case No. 8, with bilateral mastoidectomy, showed symptoms (Gradenigo's syndrome) on the left side, while the roentgenograph revealed changes in the petrous pyramid on the right.

Taylor,[26] who has worked with Kopetzky, states: "Roentgenographically the pneumatic petrous pyramid shows variations from the normal when otitic infection is present," and that "every change in the roentgenographic appearance of the petrous apex does not indicate a suppurative lesion in the petrous apex."

A single exposure showing the petrous pyramid is of little value. A progressive series of roentgenograms, beginning with the original mastoid exposure and showing an increasing density of the petrous pyramid of the affected side, is of great aid in arriving at a positive diagnosis.

Transitory Symptoms.—In the course of many of these cases symptoms of perilabyrinthitis, nystagmus, nausea and vomiting may occur. They are usually transitory, lasting only one or two days, and may be easily overlooked. They are caused by the infection passing through the cells overlying the labyrinth.

Facial weakness occurs in some cases; it is caused by an irritation of the facial nerve somewhere in its course through the petrous bone. Like the labyrinthine symptoms, it is transitory, mild in character and may pass unnoticed.

In a patient suffering from prolonged discharge following an acute otitis, accompanied by characteristic eye pain, one should always be on the alert for these less constant but significant symptoms.

Paralysis of the external rectus muscle is a relatively rare involvement in established cases of suppuration. It occurs only when the suppuration reaches the very tip cells of the bone involving the sixth nerve.

Kopetzky[24] believes that sixth nerve paralysis occurring in suppuration of the petrous pyramid is the exception rather than the rule. In two of the three cases I have had, sixth nerve paralysis occurred, but after a long period of pain, low-grade temperature and persistent discharge.

REPORT OF A CASE.

CASE 1.—M. M., white, female, 26 years old, was first seen in consultation February 3, 1934, suffering with acute purulent otitis media on the right side, associated with acute sinusitis of the same side and an upper respiratory infection of two weeks' standing. Temperature was 102° F. A myringotomy, followed by seropurulent discharge, relieved the acute pain. Two days later, some pain remained, and tenderness developed over the antral and tip regions of the mastoid. The patient was sent to the Bryn Mawr Hospital for a roentgenogram, which revealed, "a large and well pneumatized mastoid on each side. The left clear, the right

definitely hazy, and dense in the region of the antrum and mastoid tip. Definite evidence of infection with as yet no necrosis." A second roentgenogram two days later showed "an increase in density of the right mastoid, with several areas which suggest necrosis of the cell walls." This report, together with continuation of the temperature and increasing areas of tenderness and unilateral parietal headache, was sufficient evidence to warrant mastoidectomy.

At operation there was found a large area of broken down cells filled with pus occupying the space back of the antrum and in the mastoid tip. The bone was pneumatic, the cells extending into the zygoma and well down over the jugular bulb.

Except for parietal pains, which were attributed to the sinusitis, and an occasional rise of temperature to 100°, the course for the next two weeks was fairly normal. There was still some purulent discharge from the canal and mastoid wound.

During the night of the fourteenth postoperative day, severe pains in back of the eye and temple kept the patient awake in spite of large doses of morphin. The next day more pus was present in the canal and mastoid wound. The patient cried a great deal, complained of eye pain and pains in the back of the head. Roentgenogram of the mastoid showed "a few cells remaining in lower anterior portion of the mastoid and in the zygoma. No evidence of epidural abscess." Two days later mild nystagmus was noticed. It could only be seen when the eyes were turned to the extreme position. The nystagmus and some nausea lasted for three days. Examination by a neurologist was negative. Eye grounds were negative. Two days later, February 25, the neurologist and ophthalmologist reexamined the patient, with negative findings. Blood culture was reported negative.

From February 25 to March 21, the symptoms were much less severe; the temperature was normal for periods of three to six days; the discharge continued from the wound and the canal.

March 21, five weeks after the original operation, the wound was reopened. The antral region was filled with necrotic granulation tissue, but no infected cells were found. Large areas of the dura were exposed above the antrum and over the lateral sinus but were found normal. The findings were disappointing. Suppuration of the petrous pyramid was suspected.

For two or three days following, the discharge and the severity of the pains were lessened. On the third day following the second operation paralysis of the right external rectus muscle appeared, the discharge was again profuse and the pains were increasing in intensity. A roentgenogram of the base of the skull revealed definite cellular development in the petrous pyramid of the left side. On the right, the affected side, the detail was obscured, the cell walls being barely visible (Fig. 7). A diagnosis of suppuration of the petrous pyramid was made.

For ten days following there was a diminution of the severity of the pain, and the relatives of the patient would not consent to further operative procedure. On the 9th of April, twenty days after the second operation, the patient had a very bad day. Dr. F. C. Grant found no localizing evidence of brain disease. Consent for consultation with Dr. Samuel J. Kopetzky was obtained. He verified the diagnosis of suppuration of the petrous pyramid and advised operation.

The old wound was reopened and converted into a radical mastoid cavity, revealing three fistulae entering the petrous pyramid, one, with an opening 2 mm. in diameter, extending for 3 cm. into the petrous pyramid from the pretubal cells; a second, from the prebulbar cells, extending 4.5 cm. from above and in front of the superior semicircular canal; a third extended for 2 cm. into the petrous pyra-

mid. No further operation was thought necessary for drainage. Immediately following the operation iodized oil was injected into the pretubal fistula and roentgenogram showed it to reach the petrous apex.

The pains in the eye region and the external rectus paralysis gradually lessened until there was complete disappearance in two weeks. The discharge from the canal continued in decreasing amount until October 1, 1934. Since that date, the ear has been entirely dry and the patient in good health.

Bacteriologic examination revealed hemolytic streptococcus as the infecting organism throughout the course of the disease.

SUMMARY.

Suppuration of the petrous pyramid is the most recently recognized complication of purulent otitis media.

An acute case of such suppuration may resolve into a chronic purulent otitis media where there is sufficient drainage, through a fistulous opening into the middle ear or, if the cell structure is favorable, it may heal spontaneously in this manner. If drainage is not sufficient, some form of surgical assistance is necessary.

It is believed that the source of infection in many cases of chronic suppurative otitis media has its origin in the petrous pyramid.

When a radical mastoidectomy is performed for relief of chronic discharge and there is found necrotic granulation in the middle ear, especially in the region of the eustachian tube, one should suspect a fistula leading through the peritubal cell into the petrous bone.

A case typifying the symptomatology of acute suppuration of the petrous pyramid is reported.

The clinical picture of an acute suppurative petrous pyramid requiring surgical drainage is nocturnal attacks of pain in, around and back of the eye and in the temporoparietal region of the affected side, occurring with increasing intensity over a period of a few weeks, accompanying an acute purulent otitis media and mastoiditis, low grade septic temperature and evidence of progressive involvement of the petrous pyramid, as shown by a series of roentgenograms.

1910 SPRUCE ST.

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IX.

A REVIEW OF THE AURAL AND ORBITAL COMPLICATIONS OF SCARLET FEVER AT THE PHILADELPHIA HOSPITAL FOR CONTAGIOUS DISEASES FROM 1922 TO 1934.*

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Scarlet fever produces more cases of acute otitis media than any other specific disease in this country. A close second place is granted to measles. Some writers[1] would place measles above scarlet fever in their respective communities. This may be true. Certainly both are prime producers of ear trouble. The writer holds the opinion that in this community the percentage of scarlatinal otitis which proceeds to surgical mastoiditis is substantially higher than of measles.

Acute suppurative otitis media is the most important complication of scarlet fever. If we may except lymphadenitis in general and serum reaction in those patients who receive serum, we may also safely say that acute otitis media is the most frequent, as well as the most important complication of scarlet fever.

In a previous report[2], a review was given of the aural complications in scarlet fever at the Philadelphia Hospital for Contagious Diseases for the years 1922 to 1929, inclusive. It is the purpose here to bring the data up to, but excluding, 1934 in a consolidated report on otitis and to add a brief report on the orbital complications.

In this period of twelve years, 1922 to 1934, 29,298 scarlet fever patients were cared for at the hospital. Two thousand eight hundred and ninety-eight of them, or 9.9 per cent, developed acute suppurative otitis media. This may be compared with Table I, III, IV, V, which shows the incidence of otitis media in scarlet fever "in many epidemics in many parts of the world, during a period of many years, and under the observation of many different physicians."

It is interesting to note that approximately one-third of the patients who developed a running ear had a bilateral affection, for

*Presented before the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Portland, Me., January 4, 1935.

among the 2898 patients there were 4100 running ears. Four hundred fifty-five of these ears, belonging to 363 patients, proceeded to surgical mastoiditis and were subjected to simple mastoidectomy.

TABLE I.

Ears	Number of Incisions				Mastoidectomies		
	1	2	3	4	Unil.	Bil.	Deaths
Incised, 858	702	102	32	8	50	7	7
Ruptured and incised, 233	183	32	14	3	17	1	4
Ruptured, 1095					90	12	7

Number of ears	2186.0
Average number of days from operation to recovery	47.8
Number of mastoids averaged	148.0

The mortality for the mastoid patients was 35, or 9.6 per cent. The total mortality for the 2898 otitic patients was 107, or 3.7 per cent.

The average number of days from operation to recovery, meaning a healed wound and a dry ear, was 47 for 313 unselected mastoids where the period could be determined from the records.

As regards myringotomy, these 4100 ears may be divided into three groups, namely, group 1, in which there were 1852 ears which received myringotomy before the tympanic membrane ruptured; group 2, in which there were 415 ears which received myringotomy after the ear drum had ruptured; and group 3, in which there were 1843 ears in which the ear drum ruptured and did not receive myringotomy at all.

In group 1, there were 194 mastoidectomies, or 10.4 per cent. In group 2, there were 27 mastoidectomies, or 6.5 per cent. In group 3, there were 236 mastoidectomies, or 12.8 per cent.

In groups 1 and 2, where myringotomy was performed, there were 1792 ears in which the tympanic membrane was incised once, 339 in which it was incised twice, 101 in which it was incised three times, and 34 in which it was incised four times.

The writer would like to state here that as a general rule he does not favor repeated incisions of the ear drums, as he feels that the bulging of the tympanic membrane, which invites repeated incision, usually is due to the enormous swelling and edema of the mucosa of the middle ear and not due to localized pus, and repeated incision is often not only valueless but that it inflicts useless trauma on the tympanic membrane and middle ear structures.

TABLE II

Ears	Number of Incisions				Mastoidectomies		
	1	2	3	4	Unil.	Bil.	Deaths
Incised, 1852					102	45	16
Ruptured and incised, 415	1792	339	101	34	25	1	5
Ruptured, 1843					144	46	14
Number of ears							4100
Average number of days from operation to recovery							47
Number of mastoids averaged							313

About 90 per cent of otitic patients in scarlet fever are under eleven years of age. Table III gives the incidence for various age groups for those 2898 otitic patients. In the group between one and five years inclusive were 1774, or 61.2 per cent; in the group between six and ten years inclusive were 892, or 30.7 per cent; in the group between eleven and fifteen years inclusive were 135, or 4.7 per cent; in the group between sixteen and twenty years inclusive were 51, or 1.8 per cent; and in the group twenty-one years or older were 46, or 1.6 per cent.

TABLE III

Age	No. of Patients	Percentage
1-5	1744	61.2
6-10	892	30.7
11-15	135	4.7
16-20	51	1.8
21	46	1.6
Total number of patients	2898	

It has been stated by Borden,[6] with whom the writer concurs, that "it may truly be said of scarlet fever that aural complications of any degree of severity may arise at any time from the first day of acute symptoms to the last day of convalescence." This is an interesting and important practical characteristic of scarlet fever. Table IV shows the week in morbidity of the onset for the group of ears. The first week had 1021 ears; the second, 990; the third, 598; the fourth, 433; the fifth, 352; and the sixth and thereafter had 716.

TABLE IV

Week of Onset	Ears
1	1021
2	990
3	598
4	433
5	352
6	716
No. of ears	4110
No. of patients	2898

The data here presented correspond closely with those found by other writers. For example, Harries and Gillespy[7] report a series of 290 patients with scarlatinal otitis in which 40.3 per cent were under five years of age; 54.5 per cent were between five and ten; 8.3 per cent, between ten and fifteen; 2.4 per cent, between fifteen and twenty; and 3.4 per cent, over twenty.

The duration of the period of otorrhea is often annoying to both the physician and the patient. The average number of days of discharge was thirty-six for a series of 1915 consecutive ears that became dry in the hospital without mastoidectomy. This figure becomes more interesting when one considers that one usually has to decide by the fourth week whether or not a mastoidectomy is indicated.

Gardiner[8] reports that at the Edinburgh City Hospital, where no patient with scarlet fever otorrhea was allowed to leave the hospital, the average stay in the hospital of such patient was 68 days in 1919; 62 in 1920; and 52 in 1921.

In this connection it seems apropos to say something about the rôle of the pharyngeal and faucial tonsils in the development of otitis media in these patients. The nose and throat are undoubtedly the foci of infection in scarlet fever, and the presence of tonsils and adenoids contributes to the severity of the infection. If the infection reaches the middle ear, as is generally supposed, by way of the eustachian tube from the throat, then it is logical to expect that if the infection here could promptly and effectively be eradicated there would be no complicating suppurative otitis media. Unfortunately there is no method of so eradicating the infection. It was expected of scarlet fever antitoxin that it would provide to a reasonable degree such a method, but it is safe to say now that in its present efficacy it does not. On the other hand, infection may, at least in some cases, reach the middle ear by way of the blood stream. In such case it seems that an effective therapeutic method for controlling the nasopharyngeal and anginal infection would still circumvent the development of suppurative otitis media.

Among the 2898 patients here under discussion there were 169, or 5.8 per cent, who had the tonsils and adenoids removed before the development of scarlet fever. In a series of 14,733 consecutive patients with scarlet fever the tonsils and adenoids were absent in 861 patients, or 6.8 per cent.

Science has developed no great improvement in the therapeutics of acute suppurative otitis media. The useful principles of treatment

which are available today are essentially those available a quarter of a century ago. They may be outlined as follows:

(a) Prevention, by keeping the nose and pharynx free from excessive secretions.

(b) Frequent inspection of the ears, with early myringotomy in every case of bulging tympanic membrane before it ruptures.

(c) Frequent cleansing of the external auditory canal to facilitate drainage.

(d) Considering the removal of the adenoids when the ear has discharged for three weeks.

(e) Performing a simple mastoidectomy in those cases where there is persistent and profuse otorrhea, sagging of the posterior superior canal wall, nipple-shaped perforation, mastoid tenderness and edema, with fever and positive x-ray evidence of surgical mastoiditis.

ORBITAL COMPLICATIONS.

From the standpoint of the otolaryngologist, there is one outstanding important orbital complication in scarlet fever, namely, orbital cellulitis. Orbital cellulitis is most frequently due to disease of the paranasal sinuses, and this is the case in scarlet fever, with rare exceptions, such as peridental abscess or cellulitis of the face or forehead. In children, who constitute approximately 90 per cent of the scarlet fever census, it is most often due to ethmoiditis. In adults, the frontal sinus is most frequently the seat of the infection.

There are three stages to orbital cellulitis:[9] First, inflammatory edema with or without discoloration of the lids, usually most marked in the upper lid. Second, edema of the lids with exophthalmos, and with or without limitation of orbital movement. Third, the same as the second but with evidence of abscess formation.

In a series of fifty cases of orbital cellulitis, forty-five of the patients were children and five were adults. In all of them except two the orbital swelling occurred in the course of scarlet fever. In seventeen the cellulitis started in the first week of morbidity; fourteen started in the second; ten started in the third; one started in each of the fourth, fifth and sixth. Three started in the seventh; two started in the eighth and one started in the fifteenth.

In all cases except four the orbital swelling was secondary to ethmoiditis. Of the exceptions, frontal sinusitis was the focus in

two, both of whom were adults; maxillary sinusitis in one, an adult, and cellulitis of the cheek in one, a child.

In the series of fifty cases of orbital swelling, thirty-five were due to unilateral cellulitis, twenty of the right orbit and fifteen of the left; seven were due to bilateral cellulitis; six were due to unilateral abscess, four of the right orbit and two of the left; and two were due to cellulitis of the right side and abscess (in the same patient) on the left side.

TABLE V

Orbital Cellulitis Unil.		Orbital Cellulitis Bil.	Orb. Abscess		Orb. Cell. & Abs.	
Right	Left		Right	Left	Right	Left
20	15	7	11	2	2	

The modern and proper method of treating infections of the orbit has been thoroughly outlined by Porter[9] and Davis.[10] The writer's experience has been mostly with children and the treatment as applied to them in particular may be stated briefly.

The treatment depends on the stage of the disease. In the first and second stages it is conservative. X-ray of the sinuses is desirable. Ice-cold compresses are applied to the affected eye, and the nose is packed with pledgets of cotton wet with 1 per cent cocain hydrochloride or 1 per cent ephedrin sulphate, two or three times daily. The nasal fossæ must be evacuated frequently with normal saline solution or by aspirating the secretions with a mechanical suction machine and small rubber catheter, about 10 or 12 French.

When there is evidence of abscess formation or where the edema, exophthalmos and limitation of motion are marked, more radical measures are indicated.

In the milder cases removal of the anterior tip of the middle turbinate and opening of the ethmoid cells, together with an intranasal antrotomy, where there are symptoms of antral disease, will usually be sufficient. In the more severe cases ethmoidectomy by the combined external and intranasal approach is the method of choice.

The writer would like to emphasize that, according to his experience with children, the conservative method, such as outlined for the first and second stage, can nearly always be followed until the condition subsides or abscess formation occurs.

It is proper to stress the fact that an essential step in any operative procedure is to drain the sinus from which the disease spreads

to the orbit, and to caution against the inadequacy and danger of making stab incisions into the orbital tissues in order to locate a collection of pus.

SUMMARY.

Statistics concerning 2898 scarlet fever patients with acute suppurative otitis media and forty-eight scarlet fever patients with orbital cellulitis with discussions are presented.

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X.

SOME CONSIDERATIONS OF ADENOID BLEEDING.

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There are few events in the daily life of the laryngologist that cause more annoyance than the occasional telephone call, from three to six hours following an uncomplicated operation for the removal of a child's tonsils and adenoids, announcing that bright blood is trickling from the nostril or has been vomited in appreciable quantity. This event is sufficiently uncommon to warrant the feeling that it should never occur; it is potentially serious enough to brook no delay in its management; and its occurrence is often unexplainable by any facts which can be obtained by local examination. Not the least unpleasant aspect of the situation is the necessity of explanation to the parents as to why the bleeding has occurred or why it is necessary to return the child to the operating room, with or without a second anesthesia, for its control. If, in the case of adenoid bleeding, postnasal packing is deemed necessary, there is the possibility of a middle ear complication. Intravenous saline infusions may be necessary. Nevertheless, to neglect obvious active postoperative bleeding with optimistic nursing orders to apply an ice collar, give a hypodermic injection of morphin and watch the pulse is, in my experience, to put an unwarranted trust in Providence that may unexpectedly lead to a tragedy than which there is no greater in all surgery.

There may be operators who can look back over a long and busy career and fail to recall any instance of alarming postoperative adenoid bleeding, even in a large number of cases. For those men, however, who are responsible for the conduct of a large hospital clinic, in which the work must inevitably be done by a changing personnel and to some extent by a less experienced house staff, albeit under competent supervision, there will inevitably occur a certain number of cases of postoperative bleeding, requiring prompt control.

For this reason it has seemed to me valuable to make some analysis of this work in a large hospital clinic with a view to discovering if possible why and under what circumstances this complication occurs and by what means it may be lessened or entirely eradicated. I shall in the main direct the inquiry into the occurrence of bleeding

from the adenoid to such exent as requires, in the judgment of the surgeon in charge, insertion of a postnasal pack. To this end I have gathered the figures summarized in Chart I.

Chart I. In line A are summarized the figures obtained from the Out-Patient Department Tonsil and Adenoid Clinic in the Children's Hospital in Boston, for the six years, 1928 to 1934. In 10,745 operations there were listed 294 instances of postoperative bleeding, 84 from the tonsil, 210 from the adenoid, a total incidence of 2.7 per cent and of 2.0 per cent for the adenoid. It will be noted throughout the chart that in every instance, except in the comparative figures from the Manhattan Eye and Ear Infirmary, the adenoid bleeding was always more than twice as frequent as that from the tonsil. The reason for this is not at once apparent. In this series of cases the adenoid was removed last, all tonsil bleeding being entirely checked before adenoidectomy. The customary practice of suturing suspicious vessels in the tonsil fossa doubtless had something to do with this proportion. A total percentage of 2.7 per cent seems unduly high and certainly arouses one's curiosity as to its cause and its remedy. I feel that one outstanding factor was contributory. In the years 1928-1930, the clinic arrangements were such as to make it necessary for the two operators, having finished the operative work, to transfer their activities to a frequently understaffed outpatient clinic. This situation, no matter how conscientious the men, resulted in the introduction of a certain sense of pressure and haste in the operative work which I believe had a distinct bearing on the postoperative bleeding here under consideration. Each man was required to perform from six to seven operations, a small number when judged by the output of some clinics, but still a sufficient one to introduce a sense of need for completing the work as soon as possible before repairing to the outpatient clinic. No man can do his best work under these circumstances.

For this reason, modifications were made in the system to the extent that it no longer was necessary for the operators to undertake the outpatient work on the same afternoon, other help being provided for this task. The benefits of this change I believe are reflected in the figures in line B, taken for the years 1930-1934, when the improvement was inaugurated. Here the bleeding percentage in 6,145 cases is reduced from 2.7 to 2.14 per cent, a figure, however, still leaving much to be desired. During these four years the system called for operating by two distinct classes of operators: (1) a group of eleven well trained and thoroughly experienced visiting surgeons who came in on one afternoon a week, performing their six or seven operations without further clinical duties; (2) a group of twenty house

CHART I. STATISTICS OF POSTOPERATIVE TONSIL AND ADENOID BLEEDING.

Operators	Years	Number of operations	Total number of hemorrhages	Tonsil Bleeders	Adenoid Bleeders	Total percent. of bleeding	Tonsil %	Adenoid %
A. O. P. D. Clinic	1928-1934	10,745	294	84	210	2.7%	.79%	2.0%
B. O. P. D. Clinic	1930-1934	6,145	132	35	97	2.14%	.57%	1.6%
C. 20 House - Officers	1930-1934	3,116	60	13	47	1.95%	0.4%	1.5%
D. Clinic— 11 visiting men	1930-1934	3,029	72	22	50	2.37%	0.7%	1.65%
E. Private Ward (Entire staff of 30)	1928-1934	5,216	32	9	23	0.6%	.17%	.44%
F. 15 private operators	1928-1934	2,923 56%	32	9	23	1.09%	0.3%	.79%
G. 15 private operators	1928-1934	2,293 44%	0	0	0	0	0	0
H. Manhattan E. & E.	1930-1932	13,344	549	—	—	4.0%	—	—
I. Manhattan E. & E. (12 operators)	May, 1931-Nov., 1931	6,370	246	144	102	3.8%	2.2%	1.6%

officers, as adequately trained under competent supervision as possible, but naturally lacking in the years of experience possessed by the other group. At any one session a visiting man and a house officer worked side by side simultaneously. In view of this arrangement, the figures in lines C and D are most interesting. It will be noted that, in an almost equal number of operations the house officers (line C) had a total bleeding percentage of 1.95 per cent, while the more experienced visiting men (line D) had a figure of 2.37 per cent. Separate figures for tonsil and adenoid bleeding corresponded. How can this paradox be explained? Here again I believe the personal equation is the predominating factor. The attitude of these two groups is necessarily different. For the visiting surgeons, the procedure is a routine one, previously performed numberless times and to some extent a chore. To the house officer it is a relatively new experience to which he is apt to devote an intense, albeit less expert, interest. Hence, it comes about that the busier man with his outside interests and problems comes inevitably to take toward the work an attitude of a task to be performed into which creeps, unconsciously, an element of haste which is absent in the case of the less experienced man, unless he likewise becomes inoculated by the same germ through contact. Haste in this way leads, I believe, to a higher percentage of postoperative bleeding.

It becomes still more evident that this element of pressure is an important factor when we examine the record of a group of visiting surgeons in care of their own private patients. Here a man operates on one or at most two or three patients at a sitting. In many instances this group was made up of the same men working in the clinic and this affords a direct comparison between the two situations. In line E are seen the figures for the Private Ward staff of thirty men, who operated on 5,216 patients with a total bleeding percentage of only 0.6 per cent, as compared to 2.37 per cent in the clinic. Moreover, 56 per cent, or 2,923 of these cases, were operated upon by fifteen men (line F) amongst whom all the bleeding occurred, and whose percentage was thus 1.09. Two thousand two hundred ninety-three cases were operated upon by the remaining fifteen men of the private staff (line G) without any recorded postoperative bleeding whatever. How may this difference be explained? In the one instance, in which a man working both privately and in the clinic did a fairly equivalent group of operations, his bleeding percentage in the clinic was 2.0 per cent, in the private ward 1.0 per cent. In both groups the preoperative care and examination were, if anything, better in the case of the clinic patients and the etherization and postoperative care equally good. I can only fall back on the difference in mental attitude incident to the necessity of accomplishing a greater amount of work in a

given space of time. Yet even here, one may adduce figures to refute this explanation. From an analysis of the clinic figures for six years, above, it appears that the average highest monthly bleeding percentage (5 per cent) occurred in an average of 124 operations per month. The lowest corresponding percentage (0.5) was based on an average of 112 operations. In other words, there was ten times as much bleeding, although the average number of operations was only very slightly increased. This does not look as though the pressure of doing a large volume of work raised the bleeding percentage. Looked at from a different angle, it appears that the average of the largest number of cases per month (184) showed a bleeding of 2 per cent, while the average of the smallest number of cases per month (81) showed exactly the same (2 per cent). It would be hard here to maintain that the busiest months are those of the greatest postoperative bleeding. The worst monthly figure, a postoperative bleeding percentage of 6.7, was in October, 1929, the best (1.5 per cent) in November, 1932.

More recently the clinic system has again been changed, and the operating is now done entirely by the house staff, with a resident surgeon in charge. Neither of the two operators, therefore, now has any extra-hospital duties and it has been interesting and gratifying to note a still lower percentage of postoperative bleeding following this change.

For the sake of some check on these figures, I have added, in lines H and I figures from the Manhattan Eye and Ear Infirmary. In a total of 13,344 cases which, unlike our own, included adults, the bleeding percentage for two years was 4.0. A group of twelve operators (line I), working in the Manhattan Clinic for six months, had a percentage of 3.8 (2.2 for the tonsils and 1.6 for the adenoids, a reversed relation as compared with our own figures).

Granted that postoperative bleeding is to a greater or less degree an inevitable complication in a tonsil and adenoid clinic, what is its basic cause and can anything be done to avoid it? In an effort to throw some light on this question, I studied carefully certain factors in 200 clinic operations for the removal of adenoids, practically all in conjunction with tonsillectomy.

In view of the above mentioned fact that adenoid bleeding occurred more than twice as frequently as did tonsil bleeding, it is only the former which is here considered. The group of 200 was divided into two series. Series I, of 100 cases, was treated as follows. Tonsillectomy was first completed and a dry fossa assured before the

adenoid was removed. Following adenoid removal, by the use of sponge tampons in the nasopharynx, the bleeding was checked until direct inspection of the pharynx showed complete cessation of bleeding. At the same time all blood loss from the adenoid, either through the nose or saturating the sponges, was measured by weight. The weight of the adenoid and the time before the nasopharynx was found to be dry by direct inspection, were also recorded. The patient was then returned to bed and the throat inspected at hourly intervals for any signs of adenoid bleeding.

Various authors, in writing on the subject of adenoid removal, have stressed the importance of the principle here carried out in Series I, namely, the inadvisability of returning a patient to bed until the bleeding from the adenoid had entirely ceased. Surgically, this seems an obviously sound principle, comparable to a similar situation in any other part of the body. It affords opportunity, by elevation of the soft palate, either by an elevator or, as Beck advocates, with a catheter retractor, to inspect the pharyngeal wound itself and carefully search for tags and remnants, and would seem to assure the operator the greatest protection against subsequent hemorrhage. It does, however, require a somewhat more prolonged anesthesia, about which more will be said below.

In Series II an entirely different procedure was carried out. Following removal of the adenoid and before cessation of bleeding therefrom, the patient was taken back to bed, hung over the side of the latter and allowed to bleed into a basin till all bleeding ceased. This blood was likewise measured by weight as was the adenoid mass and the time until all bleeding in this position ceased.

Of these two procedures, it would seem that that of Series I was more painstaking and more likely to insure against subsequent bleeding. In series II there was no inspection of the nasopharynx beyond careful palpation for adenoid tags. The anesthesia, however, was shorter, since no additional ether was required to abolish the throat reflexes in order to permit a tranquil and complete inspection of the nasopharynx. By the same token, the patient was returned to bed in a shorter time, since there was no waiting for the immediate adenoid bleeding to cease.

This apparently somewhat lax method of Series II was adopted primarily because it seemed to be the policy of certain members of the private ward staff whose records as to postoperative bleeding were by far the best of all the visiting surgeons. It was formerly the custom of the author to assure himself of a dry nasopharynx before

any case left the operating room. A series of three bleeding cases in one week led him to inquire into the methods of other operators and to discover that this method of Series II was a routine practice with the majority of the other men. Hence its adoption as a part of this study.

In addition, all of the cases in each series had a preliminary clotting and bleeding time.

Chart II gives a general view of the results in these two series. In analyzing the individual items, a number of interesting facts come to light. The general bleeding time in the two series showed little variation. The time of operative procedure measured from the start of the tonsillectomy to the finish of the adenoidectomy varied considerably in the two series, being more than half again as long in the first as in the second. This is hard to explain, since the work was done by a group of six or seven men, whose time should average about the same. However, a large proportion of the cases was done by house officers whose technic naturally improved with experience. The average duration of the bleeding from the adenoid varied but little (four to six minutes) in spite of the different technic adopted in the two series, and, most interesting of all, the total blood loss in the two series was almost identical. There was some variation in the weight of the adenoid removed. In the first series postoperative packing was required in four instances, while in six cases after signs of bleeding the oozing subsided spontaneously. In the second series three cases required packing, while none of the others showed signs of suspicious bleeding.

It is clear from these figures that there was little to choose between these two methods, in respect to the amount of blood loss or the likelihood of postoperative bleeding severe enough to require packing. If anything, the balance is in favor of the less painstaking procedure. Can one, then, from these figures, gain any knowledge as to the type of case that is likely to give trouble or to require postoperative packing? Can one, in other words, by examination of this data, detect warnings which, if heeded, might enable one to forestall trouble?

Let us first examine the figures on those patients who required postoperative packing in the two series. The comparison between the two is shown in Chart III.

CHART II. COMPARATIVE TABLE OF 200 CASES OF ADENOIDECTOMY.

Series I. Bleeding entirely checked before patient left operating room.

Series II. Patient sent immediately to ward after adenoidectomy.

Series	General Bleeding time	Time of operation	Duration of adenoid bleeding	Adenoid weight	Weight of blood loss	Postoperative bleeding. Packed	Not packed
I.	Average 3 minutes.	Average 13 minutes.	Average 4 minutes.	Average 2.5 grams.	Average 52 grams.	4 cases	6 cases
(100 cases)	Shortest 0.5 Longest 9.0	Longest 48 min. Shortest 6 min.	Longest 20 min. Shortest 1 min.	Largest 4.6 Smallest 0.5	Largest 1529 Smallest 49		
II.	Average 2 minutes.	Average 8 minutes.	Average 6 minutes.	Average 1.5 grams.	Average 50 grams.	3	0
(100 cases)	Shortest 1.0 Longest 10.0	Longest 19 min. Shortest 5 min.	Longest 15 min. Shortest 1 min.	Largest 4.59 Smallest 0.25	Largest 1509 Smallest 89		
Total Average	2.5 minutes	12.5 minutes	5 minutes	2 grams	51 grams	Bleeding percentage, 3.5	

CHART III. BLEEDING REQUIRING POSTOPERATIVE PACKING.

Series	No. of cases	Av. operation time	Average Ad. bleed. time	Average blood loss	Systemic bleeding time (Av.)	Average weight of adenoid
I.	3	15 minutes General average 17 min.	4.1 minutes General average 4 min.	50 grams General average 52	4.3 minutes General average 3	2.2 grams General average 2.5
II.	4	10 minutes General average 8	8 minutes General average 6	80 grams General average 50	2.5 minutes General average 2.0	1.1 grams General average 1.5

These figures, alas, give little clue as to why these seven cases required postoperative packing. Beyond the somewhat excessive bleeding of 80 grams (2.6 oz.) in series II, there are no figures which differ essentially from the normal average for all the cases. So far as their statistical characteristics are concerned, these seven cases differed but little from all the others which showed no tendency to postoperative bleeding. Jones, in his report of the Manhattan Clinic, states that 90 per cent of their postoperative adenoid bleeding was checked by the removal of adenoid remains. In only two of the seven cases here summarized as requiring packing were any adenoid remains found. This fact makes it the more desirable to discover, if possible, the etiology of this complication.

With a view to determining the relationship, if any, between the various factors here recorded, certain checks were carried out between these factors:

1. Relationship between a prolonged immediate adenoid bleeding, the amount of blood involved in this bleeding and the occurrence of postoperative bleeding (requiring packing) or temporary oozing was observed. It was thought that there might be derived here something of diagnostic or prognostic value. The results are shown in Chart IV.

CHART IV. RELATIONSHIP BETWEEN PROLONGED BLEEDING, BLOOD LOSS AND POSTOPERATIVE BLEEDING.

Average of 10 longest bleeding times	General bleeding time	Average blood loss	Packed	Oozing
14.7 minutes	3.9 minutes	74.3 grams	1	1
Average for 200 cases 5.0 minutes	Average in 200 cases 2.5 minutes	Average, 200 cases 51. grams	Total in 200 cases 7	Total in 200 cases 6

From Chart IV it will be seen that in ten cases with an average immediate adenoid bleeding time three times the average for the whole group, only one case required packing and only one showed temporary oozing. In this group of ten the general bleeding time was somewhat elevated (3.9 minutes) over the general average (2.5 minutes), a very natural relationship. Also, quite naturally, the total average blood loss of 74.3 grams was considerably above the average (51 grams) and yet the postoperative complication does not appear to any real extent within this group.

2. This same situation was viewed from another angle by taking the ten largest amounts of immediate blood loss and noting their

relation to the adenoid bleeding time and the occurrence of postoperative bleeding. This is shown in Chart V.

Here again, in spite of an average blood loss twice the average for the series, there is almost no tendency for the postoperative bleeding to occur within this group. The greater blood loss corresponds, as one might expect, with the higher general bleeding time, and an increased adenoid bleeding time, but the postoperative situation fails to follow suit.

CHART V. RELATIONSHIP BETWEEN BLOOD LOSS, ADENOID BLEEDING TIME AND POSTOPERATIVE BLEEDING.

Average 10 largest blood losses	General bleeding time	Average adenoid bleeding time	Packed	Oozing
109 grams	3.3 minutes	9 minutes	1	1
Average for 200 cases 51 grams	Average for 200 cases 2.5 minutes	Average for 200 cases 5 minutes	Total in 200 cases 7	Total in 200 cases 6

3. There was a direct correlation between the size of the adenoid and the immediate blood loss attending its removal. The average weight of the ten largest adenoids, 4 grams (twice the normal average), was associated with an average blood loss of 70 grams (51 average for the 200 cases). The ten smallest adenoids (average 0.4 grams) produced an average blood loss of 33 grams. No postoperative bleeding occurred in either of these groups.

4. The question arose as to whether prolonged time of operation (and therefore of anesthesia) might have some bearing in the matter. The average of the ten longest operative times (26 minutes—average 13) was associated with an average adenoid bleeding time of seven minutes, five being the average for all the cases. No postoperative bleeding occurred in this group. On the other hand, perhaps refuting my suggestion that "haste makes waste," there were no bleeding complications among the operations done in the shortest times.

5. Finally, the ten cases showing a greatly increased general bleeding time were checked in respect to the duration of adenoid bleeding and tendency to postoperative bleeding. The average time in these ten was a bleeding time of 7.3 minutes, as against an average for the whole group of 2.5 minutes. Yet these cases showed an average adenoid bleeding time of only 6.3 minutes, 5 being the group average. Among these ten cases there occurred only one case of postoperative bleeding requiring packing.

Why, then, does postoperative bleeding occur? So far as our figures go, it is essentially independent of a prolonged general bleeding time, an unduly prolonged operation, a prolonged and increased immediate loss of blood and an excessively large adenoid. There remains one factor as yet only hinted at and incapable of definite statistical proof, which I believe may nevertheless contain the answer to the problem, namely, the depth of anesthesia. To thoroughly understand this aspect of the matter it is necessary to consider in some detail the question of anesthesia in respect to tonsillectomy and adenoidectomy.

Since the earliest history of the tonsil and adenoid operation there has been a controversy over the question of anesthesia. Before the present days of perfection in the administration of ether some of the most eminent laryngologists expressed a fear of its use and preferred to operate, at least in the younger children, without any anesthesia at all. This same, and in the modern point of view, rather inhumane attitude, has been the rule in Europe up to within the past ten years. It has been argued in behalf of this attitude that the child suffered more from the shock and fright of the administration of the anesthesia than from the pain of the operation. This can scarcely be urged today in view of the almost routinely tranquil gas-ether sequence so deftly administered by the trained anesthetist. In the year 1900 several of the most eminent members of this Society expressed themselves as in favor of adenoidectomy in children without anesthesia, even if under these circumstances it was found necessary to do the operation piecemeal at several sittings. Fatalities attributable to general anesthesia were reported with disquieting frequency. All admitted that the work without anesthesia required a deft sense of touch and dextrous manipulation of the forceps or curette, and yet we read of no reports of annoying postoperative bleeding by these men. Kimball, writing in the Transactions of this Society for 1900 on "Adenoids from the Standpoint of Hemorrhage," says that he is inclined to treat the matter as a surgical bugaboo, though it is to be noted that in twelve years he performed only 350 adenoidectomies. He decried advances in technic which hastened the time of operation and concluded that postoperative bleeding was proportionate to the rapidity of operation.

Of late years more men have advocated a complete surgical anesthesia, primarily in order that the work may be carried out with greater facility of careful inspection of the nasopharynx for any adenoid remnants. All writers agree unanimously on the point that adenoid remains predispose to postoperative bleeding. Offhand it

would seem that the more active the throat reflexes the more difficult would be the attainment of a clean pharyngeal wall, and the inspection of the adenoid bed for tags and remnants. The further objection has been offered that through muscular contractions portions of the nasopharynx, other than the adenoid tissue, might be incidentally included within the range of the adenotome. And yet, as pointed out above, the advocates of the lightest form of anesthesia—that is, none at all—seem not to have been troubled by postoperative bleeding. It may well be that the initial bleeding from the cut pharyngeal vessels is more effectively checked when the pharyngeal reflexes and muscles are more active than when they are more profoundly relaxed as in the case of a deeper anesthesia. Under the latter, the throat may appear dry when the patient leaves the operating room, but bleeding may start up again when the reflexes return. The author, for several years an advocate of complete pharyngeal relaxation during adenoidectomy, has, because of several instances of postoperative bleeding, changed to a technic which delays adenoidectomy until a complete return of the pharyngeal reflex, with a marked diminution in this complication. This same method is rigidly adhered to by several men whose bleeding percentages are the lowest found in the figures above cited. In this connection it is extremely interesting to note that in our entire series of cases, postoperative bleeding was practically unknown in those patients on whom adenoidectomy alone was performed. One wonders whether the lighter anesthesia usually incident to the shorter procedure has any bearing on this fact.

The method of removing the adenoid must be considered to have some bearing on the liability to subseqent hemorrhage. In the earliest days of laryngology it was customary to employ a curved, biting forceps (Löwenberg) which, introduced behind the soft palate, grasped the adenoid mass more or less completely and literally wrenched it from its bed. Apparently, in spite of the crudity of this procedure, subsequent bleeding was not a particularly frequent complication, though it is difficult to understand why remnants were not often left behind. Even today one occasionally sees this instrument competently used by men of the old school, somewhat to the perplexity and horror of the younger generation.

More commonly in use are two types of instruments, the curette in its various forms and the adenotome. Each has its devotees, the former particularly in the West and South. A combination of the two methods is popular, the curette being used to remove small tags of adenoid tissue left behind by the adenotome. Jones found a greater tendency to postoperative bleeding with this combination and in his

clinic at the Manhattan Eye and Ear Hospital now uses the adenotome exclusively, introducing it once in the midline, and again on either side of the nasopharynx. More recently a single instrument has been devised which combines the features of both types of instrument.

In most clinics it is customary to remove the tonsil first and then the adenoid. Some men, however, advocate preliminary removal of the adenoid, then the insertion of a gauze pack in the nasopharynx, left during the period of tonsillectomy. Jones states that this procedure has in the last 7,000 operations done at the Manhattan materially reduced the postoperative bleeding.

Various other factors must necessarily play some part. Any case of blood dyscrasia or true hemophilia or unduly long bleeding or clotting time is a dangerous case to operate upon and one in which trouble may be expected. Any technic in which the underlying muscle is invaded will be a potential case for postoperative bleeding. The presence of pharyngeal infection predisposes to similar trouble.

In the author's experience the best results and the fewest complications come from a careful preoperative investigation of the patient, the employment of a competent anesthetist, who knows at all times the depth of the anesthesia, a careful technic carried out with neither haste nor delay, and an assurance of returning pharyngeal reflexes before removal of the adenoid.

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XI.

THE EXTERNAL NOSE AS A SEAT AND SOURCE OF TROUBLE.*

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It is a practice among some barbarous nations to punish crime by cutting off the nose close to the face and then allowing the criminal to go free.

For the poor unfortunate it is a punishment worse than death, for he becomes an outcast among his own people, despised and shunned on all sides.

Why is it that whereas the loss of any other organ, as the eye, for example, excites in the beholders a sentiment of pity, the loss of the nose excites disgust and repugnance.

I wonder if it is because the nose is a peculiarly human characteristic, the one feature which more than all others distinguishes man from the beast. Standing out majestically upon the front of the face, it is something very different from the snout, the muzzle or the proboscis of the lower animals.

The size and conformation of the external nose have always been regarded as of great importance in anthropologic studies. Authorities are in agreement in accepting the so-called cephalometric index, which is the ratio of the width of the nose at its base to its length, as the most valuable single physical criterion that can be found for the classification of races.

In the science of physiognomy likewise much is made of the nose as an indication of an individual's mentality or strength of character.

In general a large, well proportioned nose is regarded as an evidence of intellectuality. Napoleon, who was a good judge of character, always looked carefully at the shape of men's noses. "When I want any head work done," he once said, "I choose a man with a long nose."

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*Read at the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Norfolk Va., January 19, 1935.

Embryologically, it is a notable fact that the external nose actually develops as an outgrowth from the brain. The nasal organ first appears as small ectodermal placodes on the under surface of the forebrain, in the region known as the nasofrontal process. Two small indentations on each side divide this process into a medial and two lateral processes. These indentations constitute the olfactory pit from which develop the nasal cavity.

At the center of the medial process there appears the so-called area of His, from which there is a downward growth which moulds itself into the formation of the dorsum and the tip of the nose.

Phrenologists perhaps will find in this anatomic fact an explanation of the nose as an index of intelligence; for must not a branch partake of the essence of its root?

As physicians we are deeply interested in the physiology as well as the pathology of the nose.

I will not go so far as to ask you to accept the theory of the external nose as an organ of ratiocination, but I do here wish seriously to protest the general tendency to regard it as a mere external appendage without useful purpose or function; or as having only the unimportant negligible function of serving as a protective covering for the organ of olfaction.

When we study the evolution of the nasal organ in the lower animals we are led to the conviction that the outer part must have developed in response to certain definite needs in connection with both respiration and olfaction.

Phylogenetically, olfaction preceded respiration as a nasal function. The external nose in its most primitive form made its appearance at the period when the latter function became grafted upon the former. In its highest development, as seen in man, it serves a purpose in properly guiding and directing the inspired air, so that it can in the most effective way subserve these combined functions.

It is now some years since it was discovered that the inspiratory air current does not take a straight course through the nasal passage but that it describes a gently upward directed curve from the anterior to the posterior nares. Paulsen, in 1882, experimenting on human cadavers, placed small strips of red litmus paper over the surface of the septum and external nasal walls, and then forced a current of ammoniated gas through the passages by means of a bellows. He found that the main current of inspired air, lifted to above the in-

ferior turbinal, passing over the region of the middle, and then descending to the choanæ.

Francke, in 1893, using a kind of smoke on prepared models, in general confirmed the findings of Paulsen, but in addition observed that the current in its passage through the fossa regularly formed at certain points definite whorls or eddies. The object evidently is to conduct the main current in a slowed rate over the respiratory region and at the same time enable portions, by slow drift and by eddies, to come most effectively in contact with the olfactory areas. It is not difficult to see that the conformation of the external nose and the size and position of the nostrils are essential determining factors in this regard. Downward directed nostrils tend to raise the height of the curve and slow the current; upward tilted or forward directed nostrils tend to lower its height and accelerate the current.

We are of the opinion that racial differences in the form of the external nose rest largely upon these principles. Inhabitants of dry cold climates have long noses with the nares in the horizontal plane, because this formation best brings about the slow, circuitous course necessary to warm and humidify the inspired air in such cases. On the other hand, inhabitants of hot moist climates have short noses with nostrils of wide dimension, and somewhat forward directed, a formation which makes for a straight current and a shorter contact with the nasal mucosa, because there is less need of the warming and humidifying functions.

The dimension of the gateway of the nasal passages can be materially modified by the action of the muscles of the external nose. There seems to be some need of recalling this fact in view of the little importance generally attached to these muscles.

The nasal muscles are of two kinds, those which act to widen the nasal opening and those whose activity is to narrow or close it. The dilator muscles, which are two in number, an anterior and a posterior dilator, run in a general vertical direction over the outside of the vestibule; while the compressor, formed of two parts, takes a general transverse direction. The posterior nasal dilator has one end attached to the maxilla, the other to the posterior part of the alar cartilage; the anterior dilator has only cartilaginous attachments. It extends from the anterior part of the lower border of the lateral cartilage to the edge of the nostril.

It may be that the chief function of these dilator muscles is merely to preserve the necessary tonus to resist the effects of external pressure on the soft parts of the nose and prevent their collapse. In

ordinary breathing the negative atmospheric pressure within the vestibule is very slight, but on forced inspiration it is decidedly increased and there is visible depression of the wings if the muscles are inefficient. The alar cartilage which constitutes the boundary of the vestibule is connected by firm fibrous tissue with the lateral cartilage above, and they move inward and outward together under the influences of the muscles. The junction of the two cartilages form an obtuse angle pointing inward toward the septum, constituting the limen vestibuli, or upper limit of the vestibule. It is a fact of considerable clinical importance that this is the narrowest part of the nasal passage, particularly that it is narrower than the anterior naris. It was accordingly referred to by Zuckerkandl as the interior naris. The upper insertion of the anterior dilator muscle is just above this line, and when the muscle contracts it acts in a way to straighten out the angle and increase the distance of both cartilages from the septum. The compressor muscle acts in an exactly contrary direction. Anyone who will take the trouble to observe this region when using a nasal speculum cannot fail to note these movements—the sphincter-like action of the compressor muscles often, in fact, proving a great hindrance to satisfactory anterior rhinoscopy.

External nose troubles arise either from diseases or from malformations. Various major and minor dermatologic affections occur not only on the outside of the nose but also within the vestibule because this has a cutaneous lining. The shape of the vestibule favors the retention of secretions within its narrow recesses. In the presence of catarrh, the vibrissæ become matted together, crusts form, and micro-organisms, especially staphylococci, are found here in abundance, in contradistinction to the fossa proper, which is normally practically sterile.

Boils, eczema and various obstinate skin affections are therefore prone to locate here, and the redness, swelling and deformity they cause, being difficult to conceal, become very embarrassing. They are often also quite painful. Tom Moore has graphically described the sad plight of the sufferer with a certain kind of external nose affection —the pimpled nose:

> "I never saw a pimpled nose
> That told of good things stowed away,
> But brought to mind convulsive throes
> That wracked the wearer night and day."

A furuncle on the external nose, as pointed out by Hurd and others, may not only be painful owing to the sparsity of connective

tissue, it may occasionally become a very grave affair. This is due to the fact that venous blood from this region is in part carried by the ethmoid vein, which communicates with the ophthalmic, whence infections may be carried directly to the intracranial sinuses.

Among the more serious affections which have their favorite seat upon the external nose are erysipelas, lupus, papilloma and rhinoscleroma. Erysipelas is due generally to streptococcic infection developing at the site of an abrasion in the vestibule. It may turn out to be a very grave infection. Papillomata of the nasal vestibule, in contradistinction to those which occur deep in the nasal fossa, are small and circumscribed, and do not have the same tendency of recurrence after removal. Lupus begins generally as a small ulcerating tumor at the anterior end of the septum. It runs a painless course with the formation of scar tissue and eventually may produce deformity of the tip of the nose. Rhinoscleroma, a disease characterized by hard, nodular granulomata in the nasal vestibule, leads to enormous overgrowth and deformity. It is fortunately rare in this country.

Malformations form a large division of external nose troubles. They may be of congenital origin as atresia; arise as results of injury, as the crooked or twisted nose; or appear simply as an exaggeration of type, as the pug nose, the long tip nose, the hump nose or the pinched nose.

The troubles which they cause are of two kinds, (1) disfigurement, (2) dysfunction. It is usually on account of the former that the surgeon is consulted, and often the trouble is more in the imagination of the patient than real.

When malformation is of a degree to constitute a deformity, plastic surgery is indicated. Not only must the surgeon who undertakes this kind of work have a very high degree of skill and originality, he must also have a keen artistic sense. In the correction, harmony of type must be scrupulously preserved. Variations apparently slight can have an unexpected profound effect upon the countenance and personality of the individual. Furthermore, the functions of the external nose muscle must be kept in mind and nothing done which will destroy or disarrange them.

There is one kind of nasal malformation which, it seems to me, has never received from the rhinologist the attention which its importance warrants. I refer to the narrow pinched nose due to alar collapse.

It is often present in young adenoid patients as a result of constant nasal obstruction and mouth breathing, and we find that it

generally disappears or greatly improves on removal of the obstructive growths. We see it also in many adults, in some of whom it is difficult to say whether it is a cause or the result of the always accompanying nasal obstruction. We find it with especial frequency in obese persons with weak, flabby muscles, and we get the impression that sometimes it is a part of the general low toned musculature.

It is easily recognized. With each inspiratory act there is a visible sinking in of the nasal wings, whereas normally they should rather expand, or at least maintain their position. With a forced inspiratory act they collapse toward the septum so that a mere chink is left at the nares. The intake of air is labored and accompanied by a sort of whistling or snorting sound or it may be entirely prevented.

To prove that the obstruction is seated, not in the nasal fossa proper, but in the external nose, all you need to do is insert the tip of a nasal speculum just within the nostril and open it so as to fully expand the boundaries of the vestibule. It is often surprising the immediate relief such patients will experience.

Students should always be taught to begin their nasal examination by observtaion of the external nose and an estimation of the functional capacity of the nares. If this precaution were more rigidly observed it is certain that patients would often be spared operations for turbinectomy and for deviated septum which are unnecessary and of course fail to relieve the complained of obstruction.

An obstruction located at the nares can cause just as much trouble as an obstruction back in the nasal fossa, and needs therefore just as badly to be corrected. If a patient comes to you complaining that he sleeps restlessly at night, and wakes up in the morning with his throat dry and parched; or if he has bad tonsils, a chronic sore throat, or an obstinate cough, with or without expectoration, do not overlook collapsed nostrils as a possible cause.

The poet Moore, who has already been quoted as an authority of external nose troubles, refers with some feeling to one of the well known effects of the too narrow, though good-looking, nose:

"I never knew a maiden face
With nostrils of the finest mould,
A nose that might a Grecian grace
But it would snore when it got old.

I never knew a charming maid,
With nostrils of the finest bore,
But when on downy bed she laid
And slept, she'd always loudly snore."

What shall we do about setting such noses right?

Plastic surgery has not been as successful here as with other kinds of nasal malformation. Attempts to widen the nostril by stripping up portions of the septum and using it as a splint to spread the alæ only have the effect of adding a new obstruction to one already present. Furthermore, any cutting operation on the wing itself is bound to be harmful to the proper functioning of the nasal muscles.

Self-retaining mechanical devices for spreading the nostrils are recommended by some authors. It is possible by a properly adjusted instrument to greatly increase the capacity of nasal respiration. Worn at night it may enable patients to rest comfortably, who would otherwise be much disturbed by laborious asthmatic-like breathing.

The chief objection to the usual instrument of this sort is, in the first place, that it cannot be worn during the day on account of appearances, and secondly, that it causes irritation by pressure against the sensitive septum used as a support.

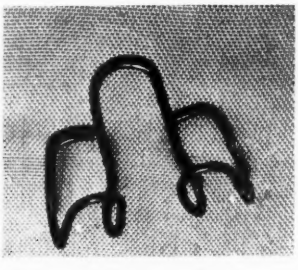

Fig. 1.

We have been employing with gratifying result a simple instrument which to a certain extent eliminates these objections. It is bilateral and so constructed that the pressure is away from the septum and against the nasal wings. A single small wire crossing the columella is barely visible.

Any mechanical device, however, no matter how well made and how competely it overcomes the stenosis, is only a temporary help.

Cannot something be done of a permanent nature, something that goes more to the root of the condition and corrects the underlying cause? We believe that much can be done, at least in those cases in which the pinched nose is due to alar collapse, as a result of partial atrophy of the nasal muscles. The obvious indication is to try to restore the tone and functional efficiency of these muscles.

It is worth remembering that all the muscles of the nose, dilators as well as compressors, are innervated by the seventh nerve, the same that governs the muscles of facial expression, and therefore presumably like them under a certain degree of voluntary control. This is not at all inconsistent with the theory that the dilator muscles function automatically under the stimulus of the respiratory act, for we see a similar combination of voluntary and involuntary functions in the laryngeal and other muscles connected with respiration.

The nasal muscles may be exercised in two ways: one by willing them to contract simultaneously with inspiration, and secondly, independent of respiration. Both are useful methods and if practiced persistently and systematically will have a decided effect in strengthening these little muscles and restoring their tonus. Practice these exercises at first in connection with deep respiration; then later without them.

You will find that, independently of the respiratory act, the muscles will contract best against a slight resistance. Place the finger gently against the side of the nostril, alternately back and front, and you can at once or at least with persistence clearly demonstrate separate contraction of the posterior and anterior muscles Subjects of nasal collapse should practice these exercises for several minutes at a time, several times a day.

If anyone argues that the very slight results to be obtained are hardly worth the efforts, the answer is that very slight results are all that are needed to make the difference between adequate and inadequate nasal respiration, and if this slight difference be bridged it means a tremendous lot to the health and well being of the patient.

It has not been our object in this brief paper to give a complete account of all the trouble that may afflict the external nose, but rather to call renewed attention of the rhinologist to a part of his specialty that seems to be relatively neglected.

It is sometimes the seat of troubles which are obviously serious; at other times, apparently slight, but important because of the serious consequences which may follow. Troubles of the external nose, therefore, despite their location, are not to be sneezed at.

We have sought to show that the external organ is not only a highly valued feature, one that is regarded as peculiarly expressive of the personality and perhaps also of the intellect of the individual, but that it besides subserves some very useful purposes in the human economy.

An organ thus valued for both appearances and utility is, of course, bound to give the owner some trouble.

But however troublesome, who could afford to be without it? To cut off the nose to spite the face would prove indeed a costly revenge. For the loss of this organ, whether by revenge or from accident or from disease, or as a punitive measure, must be accounted as a calamity of major proportions.

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XII.

LARYNGEAL AND ESOPHAGEAL ATAVISM IN MAN AS INDICATED BY THE PROBABLE PHYLOGENESIS OF THE HYPOPHARYNGEAL RECEPTACLE, CONCERNED IN THE ACT OF DEGLUTITION.*

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The following report is probably the first to be made of a particular type of laryngeal and perhaps esophageal atavism, consisting of large corniculate cartilages of Santorini attached to muscle fascicles which possibly extend up from the longitudinal coat of the esophagus. One other rare clinical case of large accessory cartilages of the human larynx, in this instance the cartilages of Wrisberg, is reproduced and recorded by Negus[7] (p. 212).

REPORT OF THE CASE.

A boy, aged 14, was referred for endoscopic examination of an esophageal obstruction. The final diagnosis, which is incidental here, was neurogenic motor dysfunction of the lower esophageal segment.† The presence of unusually prominent corniculate cartilages, separated by a very deep interarytenoid incisure, was at once of particular interest. These cartilages raised the aryepiglottic plicae high into the hypopharynx and also displayed rhythmical movements, which were synchronous with those of respiration. Upon each inspiration the corniculate cartilages were pulled postero-inferiorly; each became abducted, resulting in a wide interarytenoid incisura, and the overlying mucous membrane at this moment became puckered. On expiration, the reverse movements were seen to occur, that is, the cartilages were adducted and pulled in a vertical arc forwards and downwards.

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*From the Departments of Anatomy and Laryngology, Rhinology and Otology, College of Medicine, University of Illinois. Submitted in partial fulfillment of the requirements for the Degree of Master of Science in Anatomy in the Graduate School of the University of Illinois, 1934.

†This term is suggested by Prof. F. L. Lederer (head of Department of Laryngology, Rhinology and Otology, University of Illinois, College of Medicine) and the writer to replace in a broader sense those commonly used, such as cardiospasm, preventriculosis, achalasia, etc. Because each term in the latter group of synonyms implies a certain type of disputed pathogenesis, the one suggested should prevail until such time when distinct groups may be definitely separated therefrom.

When respiration became forced and of greater frequency, these movements increased correspondingly in amplitude and in frequency. The peculiar feature, which is still demonstrable, was that at times these events with reference to inspiration and expiration were found reversed.

Clinically, the entire act is without purpose and therefore of no physiologic significance to the patient, resulting only because of the presence of atavistic anatomic structures. The occurrence of paradoxical, or rather, of uncoordinated movements of the corniculate cartilages, is accepted by the author as additional proof of the presence of vestigeal remnants in the patient.

The signs which were observed in the patient indicate the presence of muscle fibers inserted at the most superior point of the aditus laryngis, namely, to the corniculate cartilages; these cartilages are unusual because of their large size. On the basis of the comprehensive contributions by Negus[7] on the comparative anatomy and physiology of the larynx, these cartilages and the muscles attached to them are most likely representative of atavistic forms. As will be shown, the corniculate cartilages in the patient are truly vestiges, but as for the muscle fibers, considerable question arises. It is essential that discrepancies be explained and an attempt made to define the nature of the muscle fibers, their origin and their destination.

The corniculate cartilages appear for the first time in mammals (summarized by Negus, p. 197). In fish, amphibians, reptiles and birds, the larynx is suspended in the pharynx and is so small in comparison to the wide pharyngeal cavity that there is no need for laryngeal protective mechanisms, such as are associated with a lateral group of cartilages, to keep food out of the air passages. Despite the relative increase in the size of the larynx in mammals—with certain exceptions, as the whale—the processes of respiration and deglutition may continue either simultaneously or one without the other being active. The coordination of these two acts in mammals depends upon and is assured by the addition of a number of anatomic structures, particularly accessory laryngeal cartilages and folds. The value of such adjuncts in man is evident when it is recalled that he must not only cease breathing during the act of deglutition, but that he must also resume respiration within a comparatively short period of time—the exception (Negus, p. 231) occurring in infants in whom liquids may be received by the esophagus without the necessity of interrupting respiration.

Not only are there the wide variations of protective mechanisms, as already indicated, between man and other species, but there occur, too, between man and different mammalian types definite divergences

in the laryngeal, pharyngeal and esophageal structures and in the acts of deglutition and respiration. The differences (Negus, p. 182) have been evolved mainly because of dietary habits, and the consequent need for modifying the relation of the air to food passages, as we see it contrasted, principally in *herbivora:* deer, antelopes, monotremes, marsupials (except Tasmanian devil and Tasmanian wolf, Negus, p. 194), llamas, horses, paradoxures, squirrels, marmots, hares, rabbits, oxen, gazelles, lemurs, bats, bisons, gnus, giraffes, rats, hedgehogs, moles, sheep, goats, etc. (p. 184), and *carnivora,* cat tribe, hyenas, mongooses, civets, cheetahs, aard wolves, bunturongs, etc. (Negus, p. 184).

In the lower types of herbivorous animals, palatine and epiglottic folds perform the service of guides, as it were, to direct foods into the proper channels and prevent aspiration into the trachea. They need only to be mentioned here. A higher type of laryngeal specialization is encountered in the ruminants (Negus, p. 200) and other animals which chew grass and vegetable foods. This consists of an increase in height of the arytenoid folds, attained by the elevation not only of the posterior end of the whole larynx itself at the level of the arytenoid eminences, but also of the membranous prolongation of the glottic margins, namely, the thyro-arytenoid folds. The result (Negus, p. 201) is that the margins of the glottis are raised above the level of the floor of the pharynx in the whole of their length and that a deep channel is left on either side of the larynx, irrespective of that formed by the lateral epiglottic folds; along this additional lateral channel, created by the larynx, liquids may travel without the danger of inundating the glottis even when it is open. It may be assumed, on the basis of such physiologic premises, that the posterior end of the larynx is more efficiently raised for herbivores by the addition to the arytenoid cartilages of two bodies, the corniculate cartilages. Infants retain such lateral food channels—indicated in the patient—which are lost early in life; the loss is coincident with the developmental displacement of the larynx into lower levels of the neck and consequent removal of the vestibule and epiglottis, that are relatively high early in life, from the proximity of the soft palate.

The cartilages of Wrisberg, probably for the same reasons, are differentiated from, though still linked with, the epiglottis (Negus, pp. 209-210), as is to be found in the larynx of the great ant eater and tamandua; they become separate structures in the badger, otter and panda, and more so in the spider monkey, woolly monkey and the marmoset.

The foregoing facts show definitely a phylogenetic differentiation and development from the epiglottis of the accessory cartilages of the larynx.

In most of the *carnivora,* on the contrary, the lateral plicæ are not well marked (Negus, p. 211), and since the aryepiglottic folds are very low and the lateral palatine folds are short, there is no lateral food channel worth mentioning. Such is not needed, evidently, as the diet consists mostly of lumps of flesh which are swallowed whole. In man (omnivorous) the aryepiglottic folds are so low as to be very inefficient in forming a good lateral food channel; nor are there any long palatine folds to act in association. In the carnivorous and omnivorous animals the larynx has to be closed for swallowing in the same manner, both for liquids and solids; a mechanism is called for to permit rapid transit of food through the oropharynx and into the esophagus without the food being displaced into the larynx.

The attachment of the anterior wall of the esophageal orifice to the larynx in man, and the rising of the latter during the act of swallowing provide a hypopharyngeal receptacle, which at once receives material propelled into it by the tongue.[5] According to Negus, in the greater number of carnivorous animals this mechanism is accomplished by the attachment of the longitudinal muscle fibers of the anterior portion of the esophagus to the tips of the corniculate cartilages in the manner of a sling. It is in connection with the description of this that Negus uses the term "double mechanism" to designate what he considers a perfect arrangement between the upper respiratory and the upper gustatory tracts. His analysis of the individual coordinated movements of this mechanism reveals that they consist of (Negus, p. 196): (1) the contraction of the interarytenoid in association with the thyro-arytenoid muscles in a sphincteric manner; (2) because of this associated action and the position of the interarytenoid muscles below the arching cartilages of Santorini, the muscles in their contraction pull the hooklike processes forward nearer to the thyroid cartilages; (3) it happens, then, because of this rotation together with the slight movement of the cricoid, that their posterior processes or tips are tilted upwards; (4) when they do so, they pull up the top of the esophagus, because of the laxness of the underlying connective tissue and the attachment of the esophagus by fascial fibers (in the kangaroo) to the tips of these cartilages, and draw it forward toward the thyroid cartilage; (5) consequently the food passage is made to open in a funnel-like manner—the hypopharyngeal receptacle of the writer—at the moment when the laryngeal aperture is closed.

The great ant eater has short corniculate cartilages and never swallows a bulky bolus; squirrels and sloths have very small ones; whales, in which the size of food is small in proportion to the caliber of the gullet, have none. In contrast to these examples, which illustrate the direct relationship between diets of small caliber and small or absent corniculate cartilages, it is interesting to note that "In dogs . . . not only are there certain aponeurotic fibers of the esophagus attached to the processes of Santorini, but that some longitudinal muscle fibers from the esophagus are directly continuous with the thyro-arytenoid muscle. This arrangement must give material aid to the mechanism described, by which the esophagus is opened when the larynx is closed" (Negus, p. 223), coinciding with the bulky character of the diet. The writer has found, as has Negus, the same anatomic peculiarity in a number of gross dissections of the human larynx. In man (Negus, p. 219) there is attachment of the esophagus to a considerable extent of the posterior or dorsal wall of the cricoid cartilage; "the result is that the striking double mechanism is not nearly so efficient in the human species as in other types."

The fact that herbivorous animals, such as the deer, antelope, llamas and horses, have well developed cartilages of Santorini not only accounts for the ability of these animals to go on breathing and eating at the same time—so that the juices of grass or small quantities of chewed-up herbage pass along the lateral food channels while the glottis is widely open—but also provides, with the attachment of the esophagus to those cartilages, hence, a carnivore specialization, a good mechanism for times when it is necessary for them to swallow a large bolus.

The structures which exist in the patient's larynx and hypopharynx appear to represent a similar combination in its vestigeal or atavistic form (Fig. 2).

From the preceding extracts taken from Negus' book, it is certain that he has provided the laryngologist with the first description, in his studies of comparative anatomy and physiology of the larynx, of a double mechanism which the laryngologist has not fully recognized or appreciated heretofore. The writer had made similar observations independently, but interpreted them differently, particularly in his report[3] of a newly determined anatomico-physiologic mechanism in the pathogenesis of postcricoid foreign bodies. Both the author* and Negus† called attention to the failure of many writers

*Jackson, Haslinger, Waldapfel, Wiethe, Abel, Birmingham, Cunningham, Howell, Kronecker and Meltzer, Cannon and Moser, listed in the writer's paper of 1933.

†Symington, Birmingham, Abel, listed in Negus, p. 219.

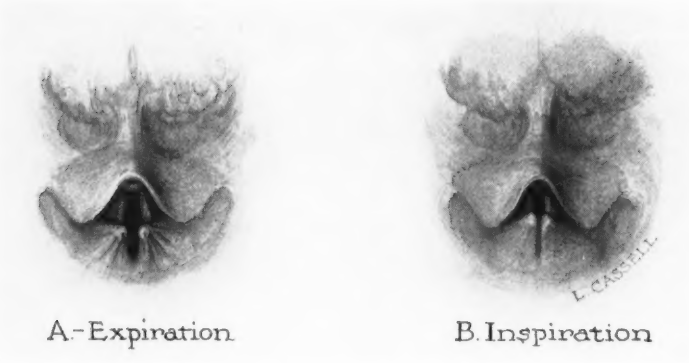

Fig. 1. Drawings of laryngoscopic views of the larynx of the patient during expiration (*A*) and inspiration (*B*).

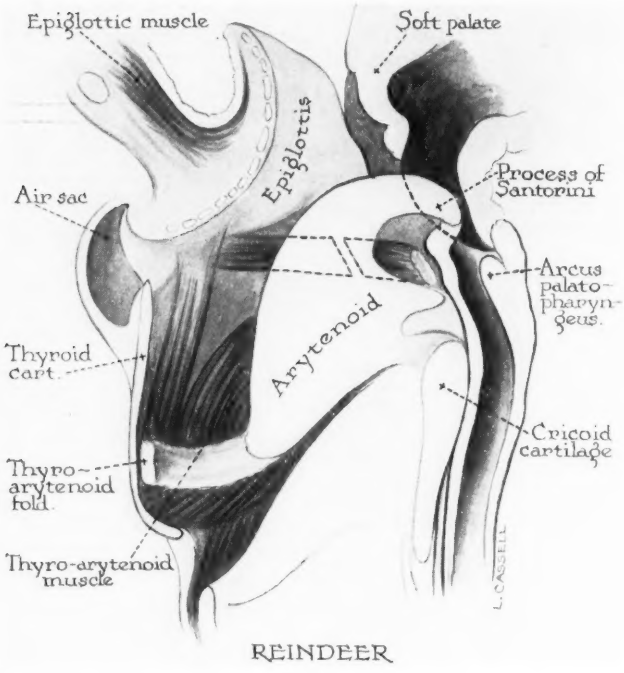

Fig. 2. Drawing reproduced from Fig. 77, p. 196, Negus.

on the anatomy of the esophagus and larynx and the physiology of deglutition, to advance any functional significance to the structures which we consider to be special anatomic provisions for the act of swallowing. But, as already suggested, there are—as far as the human organism is concerned—a number of discrepancies between the interpretation by Negus of a double mechanism and by the writer of a hypopharyngeal receptacle.* Negus (p. 219) describes the attachment of the esophagus as a whole to the corniculate cartilages, whereas the present writer has stressed—on the basis of the usual findings in man—the attachment of definite parts of the esophagus to the cricoid plate. Furthermore, a decided difference exists between the implication of a "double mechanism," which depends upon movements of the corniculate cartilages, as has already been reviewed, and the mechanism relating to the formation of a hypopharyngeal receptacle. The latter occurs as the result of the fixation of the hypopharynx to the cricoid plate which is pulled upward with the larynx during the first phase of the second stage of deglutition. It does not depend upon any action of the thyro-arytenoid muscles, interarytenoid muscles or corniculate cartilages which effect glottic closure, but is a distinctly separate mechanism that provides rapid transit of a bolus into the lower levels of the pharynx.

The writer's dissections permit additional observations on this mechanism which was briefly reported earlier. The accompanying photograph of an anatomic specimen (Fig. 3) and the drawings (Fig. 4) made from it and from other dissections of the particular structures, give a definite impression that the longitudinal muscle coat of the esophagus (*E*) is continuous with a stout tendinous band (*C*), which is firmly attached to the superior border of the cricoid plate (*B*) and loosely connected with its inferior border by areolar tissue (*D*). The figures in textbooks, showing this tendinous band, portray its specific features inadequately, although Birmingham as early as 1898 expresses surprise at the fact "that the esophagus can be easily separated from the back of the cricoid almost as far as its superior border, displaying the posterior crico-arytenoid muscle freely, while leaving the esophagus intact." At the superior attachment of this tendinous band (*B*), only sharp dissection can separate it from the perichondrial fibrous tissue of the cricoid cartilage.

Springing from the superior border of the cricoid cartilage, a second band (*A*), not as heavy and distinct as that below, runs

*Barclay ('30, '32) has also noted this peculiar "open-mouthed receptacle of the Christmas stocking type"; the radiologic interpretations relating to this receptacle were not analyzed anatomically.

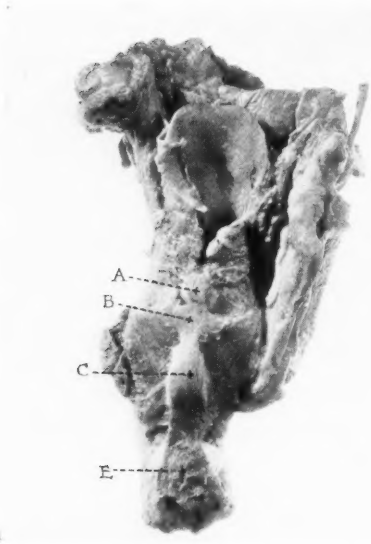

Fig. 3. Photograph of posterior view of larynx from a cadaver, showing the longitudinal muscles *(E)* of the anterior wall of the esophagus extending to the level of the cricothyroid articulation and merging with the tendinous band, *(C)*, which is attached to superior margin of the cricoid cartilage at *(B)*. At *(A)* a second fascial thickening.

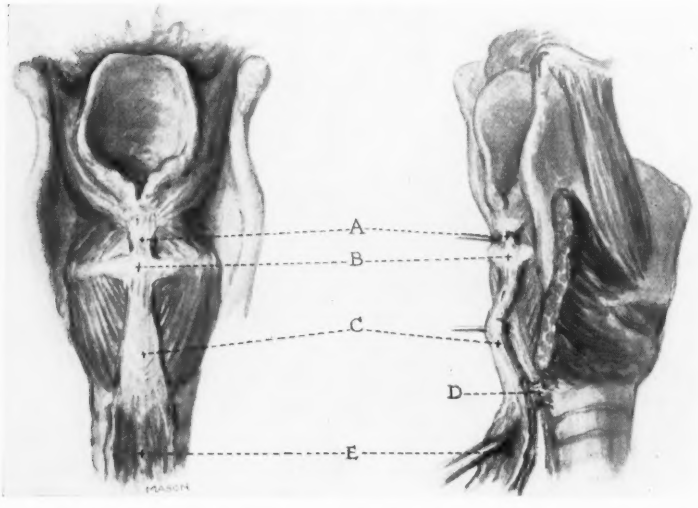

Fig. 4. Drawings of dissection of larynx, shown in dorsal and lateral views. Refer to Fig. 3 for explanations. *D*, loose connective tissue attachment of upper end of esophagus to lower part of cricoid cartilage.

upwards and appears to merge with the fascia of the interarytenoid muscle. Though this band probably is a factor in the mechanism of phonation and in the sequelae to laryngeal paralyses, it plays no rôle in deglutition. Traction on the esophagus produces absolutely no effect either upon the superior band (*A*), the adjacent corniculate or the arytenoid cartilages; the firm union with the superior border of the cricoid plate prevents any such possible transmitted action from below.

Secondly, the mucous membrane of the hypopharynx, as it continues from below to envelope the laryngeal structures, is well known to be extremely lax over these parts because of the areolar character of the underlying submucous tissues and fascia. Traction cephalad, or traction ventrad on the cartilages of Santorini has a minimal effect, consequently, upon the loosely fixed mucosa of the hypopharynx, resulting merely in a stretching of it.

From the foregoing description, it requires but little physiologic detail to demonstrate that any stress or strain applied through the cartilages of Santorini in man can have scant or no effect on the anteroposterior diameter of the hypopharynx. On the other hand, any displacement of the larynx forward and upward must have a marked effect on the hypopharynx by pulling its anterior wall along with the larynx, thereby increasing the anteroposterior diameter of this region and definitely opening it up. A "hypopharyngeal receptacle" is formed which is the antithesis of the obliterated space seen in the state of tonic contraction during respiration.

In lower types of animals the double mechanism deduced by Negus on the basis of his observations of what he considers to be consistent attachments of the esophagus to the processes of the corniculate cartilages—an arrangement which is questioned by the writer—would be an evident one, and no doubt would assist materially in the act of swallowing large or small masses of food. The same arrangement certainly does not exist in the usual specimen of the human larynx. The picture (No. 5), which is reproduced from Negus' book, illustrates his anatomic conception of the parts representative in man. The writer again questions the morphologic structure of these attachments. If they are actual tendinous continuations of the longitudinal muscle of the esophagus to the corniculate cartilages, they represent anomalous vestiges; if they are merely reflections of the pharyngobasillar fascia, traction on them during deglutition can have only minimal effects upon the hypopharynx and the esophageal orifice. The foregoing studies of the characteristic attachments of the esopha-

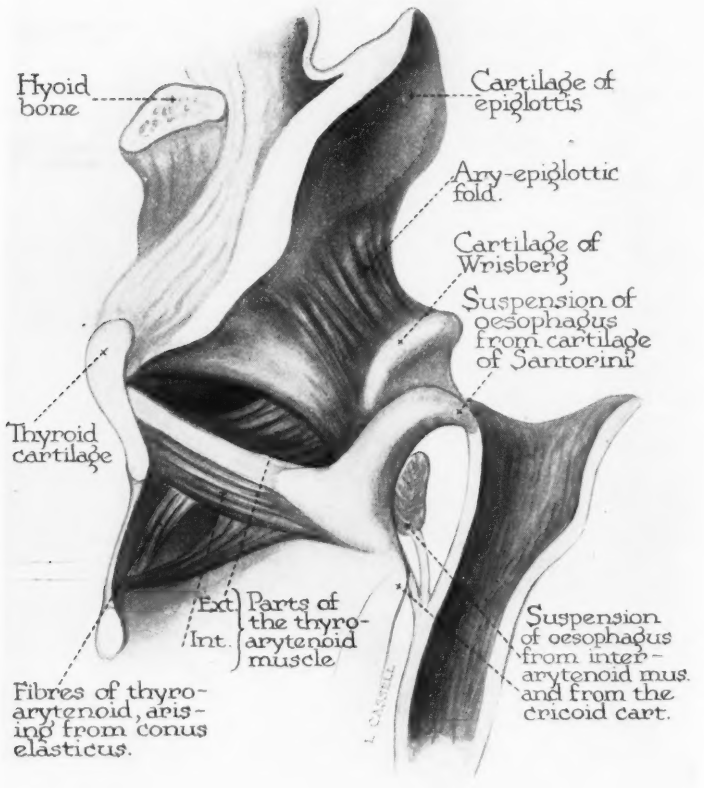
Hyoid bone
Cartilage of epiglottis
Ary-epiglottic fold.
Cartilage of Wrisberg
Suspension of oesophagus from cartilage of Santorini
Thyroid cartilage
Ext.
Int.
Parts of the thyro-arytenoid muscle
Suspension of oesophagus from inter-arytenoid mus. and from the cricoid cart.
Fibres of thyro-arytenoid, arising from conus elasticus.

Fig. 5. Drawing reproduced from Fig. 97, p. 219, Negus.

gus to the larynx and of the laxness of the underlying areolar fasciæ has established these alternatives.

In concluding that phase of Negus' work, upon which he bases his reasons for assuming an inefficient double mechanism in man, the following quotations leave much that is doubtful in this reader's mind: "Man is well off, although in his case the esophagus gains some attachment of the interarytenoid muscle and to the top of the posterior surface of the cricoid plate, as well as to the tips of the cartilages of Santorini" (p. 224). Again, "In man (p. 229) the esophagus is attached in part to the tips of the cartilages of Santorini, but there is in addition a considerable

degree of suspension from the back of the cricoid ring." Finally, he contradicts his statements mentioned earlier (p. 219) when he concludes that "The function of the cartilages of Santorini is to open the mouth of the esophagus when the laryngeal aperture is closed. This action is attainable because the aponeurosis of the esophagus is suspended from the tips of the cartilages; furthermore, some fibers of the longitudinal muscle of the esophagus are continuous with the interarytenoid and thyro-arytenoid muscles" (p. 454); and "In man the cartilages are still long, but do not give major suspension to the esophagus; the latter is attached in a considerable degree to the back of the cricoid cartilage in a manner quite dissimilar to the case of most other animals" (p. 477).

To evaluate further the discrepancies between the gross anatomic accounts of Negus and the writer, it was decided to prepare microscopic serial sections* of the structures in question of a fetus, since they might yield phylogenetic as well as gross anatomic data. If, as Negus states, the esophagus is attached to the tips of the corniculate cartilages in certain animal types, as discussed earlier in this paper, it was thought that fascicles from the muscular layer of the esophagus which may have degenerated during postnatal life would be found in anatomic specimens of the human fetus in keeping with Müller's and Haeckel's law of biogenesis.. Because the circular muscle layer of the esophagus in the human fetus is usually indicated at 10 mm. and the longitudinal at 55 mm., the fetus of 170 mm. (twenty weeks) was judged to be sufficiently advanced in development to show all muscle tissue that is laid down.

Photomicrographs (Figs. 6, 7 and 8) show the longitudinal muscles of the upper segment of the esophagus to be arranged in bundles of varying dimensions. At the level of the upper three tracheal rings (Fig. 6) there is a scarcity of longitudinal muscle fibers in the medial portion of the anterior and posterior walls of the esophagus; as the lateral border of the anterior wall is approached the muscle fascicles increase in number and are fairly large within the extreme lateral portions of the esophagus. This structural peculiarity was demonstrated grossly by Birmingham[3] in the adult cadavers and is also a striking feature in the writer's microscopic series from the 170 mm. fetus. The agreement between these findings in the adult and fetus substantiates the interpretation of gross anatomic structures in the adult on the basis of histologic determinations in a fetus.

*The preparation of a larynx, trachea and corresponding levels of the hypopharynx and esophagus of the 20 weeks (170 mm.), fetus was mounted in paraffin and cut in thicknesses of 25 micra each; the stain used was hematoxylin-eosin.

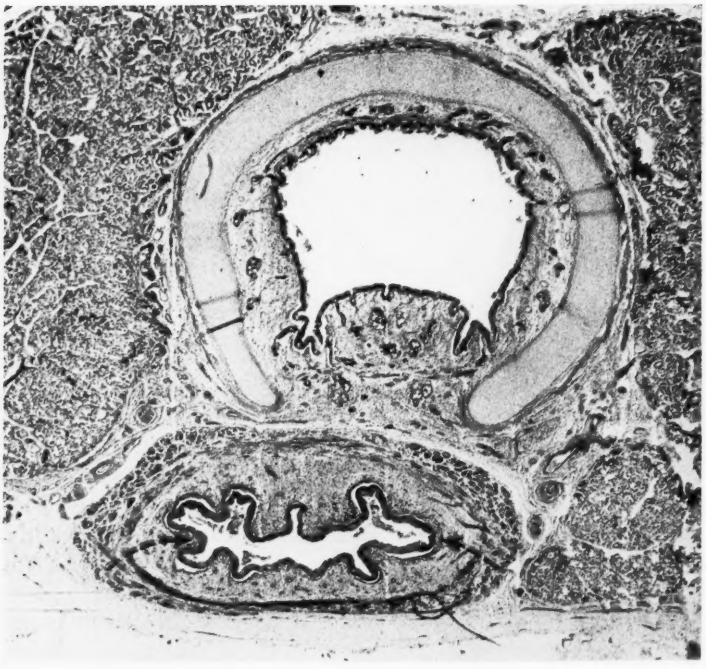

Fig. 6. Photomicrograph of a transverse section through the second tracheal ring and esophagus from a human fetus of 170 mm. (5 months) × 17. Note the scarcity of longitudinal muscle fascicles in the anterior and posterior walls of the esophagus, and their abundance in the lateral walls. Arrows point to muscularis mucosae.

If any question exists concerning the possible demarcation of additional muscles later, the similarity between the muscular grouping, as described by Birmingham in the adult and as observed by the writer in the 170 mm. fetus, establishes the basis for considering the other observations on the larynx, esophagus and hypopharynx in the fetus to be valid for the adult.

As the level of the cricoid plate is approached, the median fascicles disappear, and the lateral ones turn anteriorly and somewhat medially. At the level of the cricothyroid articulation (Fig. 7) all of the longitudinal muscle fibers of the esophagus are absent, likewise the circular ones, being replaced in the anterior wall of the hypopharynx by the continuation of the pharyngobasilar fascia, and in the anterolateral and the posterior walls by the fascicles of the inferior constrictor muscle (cricopharyngeal constrictor) of the pharynx.

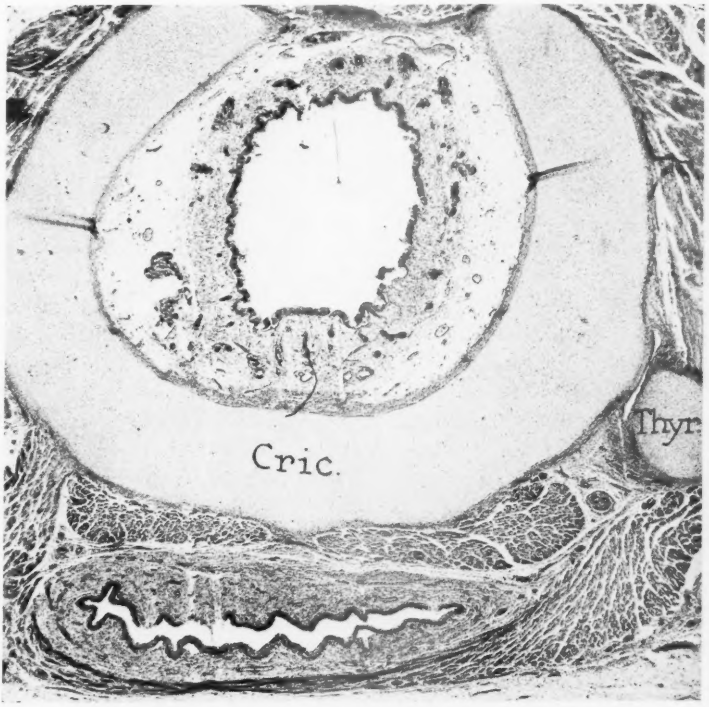

Fig. 7. Photomicrograph of a transverse section through the larynx at the level of the cricothyroid articulation from a human fetus of 170 mm. (5 months) × 19. *Cric.*, cricoid plate; *thyr.*, inferior cornu of the thyroid cartilage. Note the absence of muscle in the anterior and antero-lateral walls of the hypopharynx. The muscularis mucosae seen in Fig. 6 has disappeared.

The findings merely confirm the fact that the esophagus *per se* does not exist at this level, and that it is erroneous to refer to that segment of the gustatory tract at the level of the larynx as esophagus. But, because there exists a transition between the various walls of the esophageal orifice and those of the lowest level of the hypopharynx, the implication of esophageal attachments to structures above it is to a certain extent permissible. Anatomically the submucous coat of the esophagus is considered to be continuous with the pharyngobasilar fascia above, the difference between these layers at the two levels being the absence of muscularis mucosæ in the pharynx. Since none of the sections show the muscle fibers of the esophagus as arising from or inserting into the perichondrium of the cricoid plate (Fig. 8), the only means whereby they must attach in the region of the cricoid

plate is through the submucous layer of the anterior wall of the hypopharynx, perhaps by the rich layer of longitudinal elastic fibers found in the submucosa of the pharynx.

It has been shown by Birmingham (1898) and generally accepted, as evidenced in various textbooks and reports (Quain,[9] Spalteholtz,[10] Abel,[1] Negus,[7] Cunningham,[4] Fishman[5]) that there exists an attachment of the fascial layer of the hypopharynx to the cricoid plate. Microscopic studies show that there is no origin or insertion, in the strict sense of the words, nor is there any "stout tendon," as we all have assumed from our gross anatomic dissections. The structure which we designate as the esophagus ceases to exist, with the changes in the histologic structure which take place, at about the level of the cricothyroid joint to become instead that part of the gustatory passage termed the hypopharynx. True, the submucosa of the esophagus and the adjacent fibrous tissue, comprising the pharyngobasilar fascia of its anterior wall, continue over the cricoid plate as the connective tissue floor of the hypopharyngeal epithelium. What then is the manner of attachment so definitely demarcated by blunt dissection? The microscopic sections of this region, represented by the photomicrograph of Fig. 8 show no definite merging with the perichondrium. But there is a marked thickening of the fascial layers corresponding somewhat to a band or tendon, though not as sharply defined. It is this connective tissue that is shown as a band in the dissection and drawings from it (Figs. 3 and 4). This structure appears to have an attachment, more or less distinct, to the perichondrium of the superior border of the cricoid plate; farther up in the region of the corniculate cartilage not even the suggestion of such an attachment is seen.

Throughout his stimulating studies in comparative anatomy and physiology of the larynx, Negus wishes the reader to think in terms of loss and displacement to various levels of the head and neck of particular structures essential for certain acts as the animal types develop and acquire new habits and environments. In connection with the attachments of the esophagus, interpreted in terms of mechanisms for deglutition, he (p. 477) comes to the philosophic deduction that "in early man there was not the same attachment of the esophagus to the cricoid, but rather more free suspension from the tips of the cartilages of Santorini." On the basis of such authoritative premises the writer assumed that the large corniculate cartilages in the clinical case and the muscle fibers that produce movements of these cartilages and puckering of the mucosa both represented atavistic structures.

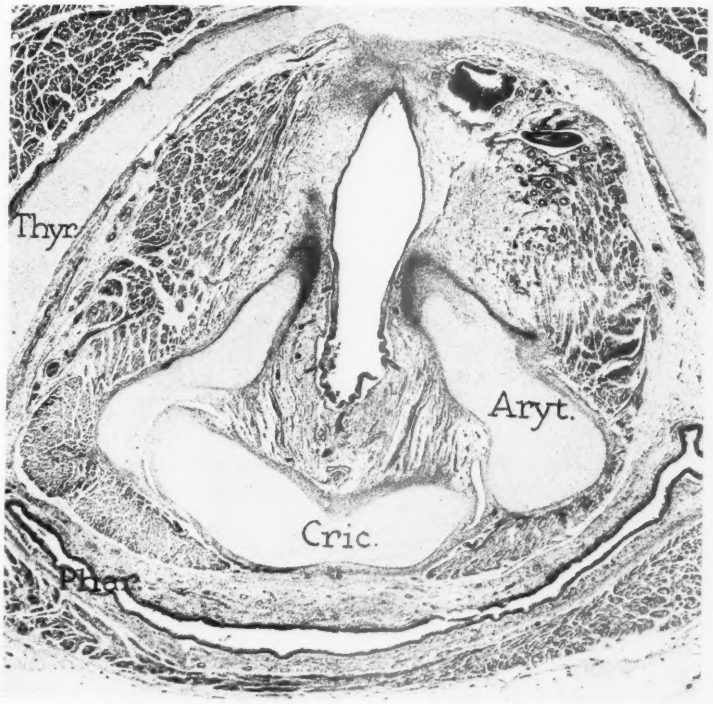

Fig. 8. Photomicrograph of a transverse section through the larynx near the upper margin of the cricoid plate (*cric.*); *aryt.*, arytenoid cartilage; *thyr.*, thyroid cartilage; *phar.*, hypopharynx. Note the thickening of relatively dense connective tissue in the anterior wall of the hypopharynx adjoining the middle portion of the posterior surface of the cricoid plate; laterally the connective tissue is seen to be of looser texture. Human fetus of 170 mm. (5 months) × 16.

Regarding the latter, it has already been established that the usual attachment of the esophagus is a relative one to the hypopharyngeal fascia, and that no esophageal muscle fibers were found at a level higher than the cricothyroid joint. What then may the basis be for assuming, if these structures are to be called atavistic muscle fibers of the esophagus, that the contractions described were indeed produced by muscle fascicles of the esophagus inserting into the corniculate cartilages? Were the writer to accept the descriptions by Negus, this question need not be propounded, and searches of additional embryonal and adult specimens could be continued for the purpose of finding esophageal muscles at such high levels in the gustatory tract as the laryngopharynx. According to Oppel,[5] these

searches should be successful, for he would have the striated muscle fibers of the esophagus derived from the pharyngeal constrictors as a result of the downgrowth of the latter upon the esophagus. The ultimate derivation of esophageal vestiges would then be from branchial muscle which was regarded by most investigators, according to McGill,[6] as arising from the lower myotomes of the head.

These views would coincide well with those of Negus and would permit the use of the term "esophageal atavism." However, the use of the word esophagus for hypopharynx by Negus in describing his gross specimens has caused considerable hesitation on the part of Professor Kampmeier to accept the statements of Negus relating to the evolution of these muscle fibers of the esophagus. The cause for doubt is the fact that Negus has failed to establish definitely the unquestionable muscular nature of the tissues under discussion which constitute the longitudinal bands from the esophagus and also their modes of attachment to the cartilages of the larynx. Our doubt is augmented by the histologic studies in pig and dogfish by McGill,[6] who demonstrated that the striated and smooth muscle cells of the esophagus have a common derivation from the mesenchymal syncitium, the striated cells being a further differentiation of smooth muscle cells; that there is no downgrowth apparent from the pharyngeal region; and that there is no connection at any stage of development between the mesenchymal muscle-forming tissue of the esophagus and the cells of the myotomes.

The writer's final argument against his use of the term atavism with reference to the esophagus might be McGill's statement that the striated muscle cells of the esophagus during its histogenesis do not show degeneration associated with the invasion of fibrous tissue as do skeletal muscle cells. It may be stated, accordingly, that the muscular arrangement in the esophagus, as found in the adult, is the result of early development in utero rather than postnatal degeneration. If this is correct, we may add on the basis of our studies in a 170 mm. fetus that there can be no attachment of the longitudinal muscle fascicles of the esophagus to the tips of the corniculate cartilages at any time in the normal development of man. It follows that degeneration and secondary attachment to lower levels of the gustatory tract, as claimed by Negus, cannot take place. Atavism is then a misconception and the term anomaly is more appropriate.

Because histologic studies are not possible of the structures which produce these movements of the corniculate cartilages in the patient, any interpretation of these movements can only be conjectural. Positive evidence of their atavistic character will be furnished when

definite attachments of muscle bundles of the esophagus to the cricoid plate and corniculate cartilages are shown histologically to occur occasionally, as the writer's case would tend to indicate; otherwise these muscle fibers can represent but one situation, namely, anomalous muscularis mucosæ within the hypopharynx. The latter condition is suggested by Professor Kampmeier as the more probable interpretation of the two on the basis that the movements of the corniculate cartilages were slow in character, corresponding to the rhythmic contractions of smooth muscle rather than to the short, quick jerks of striated muscle.

SUMMARY.

1. (a) On the basis of deductions from anatomy, the primary function of large cartilages of Santorini is to increase the height of the aryepiglottic plicæ, thereby providing another type of lateral food channel.

(b) This channel—found characteristically in herbivorous types, though present to a lesser degree in carnivorous mammals—provides a mechanism which apparently is adapted to the action of swallowing liquids while respiration continues uninterrupted; infants retain such a mechanism.

(c) The unusually large cartilages of Santorini which were present in the patient are, therefore, phylogenetic vestiges, and are the first to be reported in man as a form of laryngeal atavism.

2. (a) A morphologic study, both gross and microscopic in man, of the relation of the esophagus to the cricoid cartilage and to the hyopharynx shows that the hypopharyngeal submucosa and adjoining reflections of the pharyngobasilar fascia are loosely applied over the pharyngolarynx, but are more firmly fixed to the superior and medial borders of the cricoid plate. In the fetus the character of the submucosa overlying the cartilages of Santorini and Wrisberg is entirely loose and areolar; it is more dense in the adult specimen.

(b) It is erroneous anatomically to speak of direct attachment of the esophagus to the cricoid plate, because the band of fibrous tissue which is attached to the upper middle margin of the cricoid plate is a part of the pharyngobasilar fascia and the submucosa of the hypopharynx.

(c) Laryngeal effects by traction directly upon the esophagus are proven to be nonexistent in man, contrary to the suggestions by Negus; they are, instead, effects upon the hypopharynx. The latter gives material assistance to deglutition in the formation of a "hypo-

pharyngeal receptacle," adapted for the purpose of swallowing large masses of food, and, according to Negus, found characteristically in carnivorous types, though also present in many herbivorous mammals.

(b) The origin of the muscle fibers which produced the contractions of the large corniculate cartilages in the patient remains undetermined. It is possible that such fibers may represent either phylogenetic vestiges of longitudinal striated muscle fibers of the esophagus or an anomalous presence at the level of the pharyngolarynx of a muscularis mucosæ.

3. There is no provision for a "double mechanism" in man, since it has been shown anatomically that only the rise of the larynx accompanying deglutition can produce a "hypopharyngeal receptacle." This mechanism is well adapted to man's omnivorous diet.

The writer wishes to acknowledge his appreciation of the inestimable value of the constructive criticism of Professor Otto F. Kampmeier, Head of the Department of Anatomy, University of Illinois College of Medicine, throughout the progress of this work. Also thanks to Professor Francis L. Lederer for his enthusiastic support.

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XIII.

HEMORRHAGE IN LUNG CANCER: A FATAL CASE FOLLOWING BRONCHOSCOPY.*

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CHICAGO.

The present trend of diagnostic and therapeutic endeavor is fast placing the bronchoscopic efforts of the laryngologist in the foreground in bronchiogenic carcinoma. His aid is sought in all questionable cases which clinically and roentgenologically have been obscure. Inasmuch as this form of malignancy has grown statistically to a formidable 10 per cent of all cancers, greater attention is being focused on its recognition, and bronchoscopy has played no small part in making possible the premortem diagnoses, and in a few instances, the early findings of a lesion which responded to therapy.

Diagnostically the consideration of this disease has been greatly altered since the beginning of the century, when only 5 per cent were recognized, i. e., 95 per cent were diagnosed at autopsy. Now it is felt that with the roentgen observation, together with bronchoscopy, there should be 90 per cent of positive diagnoses, the latter method, especially if Jackson is correct, is stating that 85 per cent of the lesions originate in the bronchial mucosa. In these instances a positive biopsy should be quite simple, although from the literature it would seem that the percentage of biopsies does not indicate this high degree of accessible neoplastic material. Of course, other findings, such as broadening of the carina, fixity of the tracheobronchial tree and distortion (kinking, deflection and stenosis) may be almost pathognomonic but leave some doubt. The postmortem table in these instances is after all more conclusive. The bronchoscopic observation in such cases then expresses merely a tentative opinion as to the possible existence of a neoplasm in the surroundings of the bronchus.

The symptoms of the disease are variable, depending upon the stage of the disease and the site of the lesion (endobronchial, peri-

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bronchial or parenchymal). The majority of these patients give a definite history of some pulmonary episode, such as pleurisy, pneumonia, etc. There is, of course, a cough and wheezing, which is heard at the open mouth. This wheeze, according to the Jacksons,[1] is one of the very important and most frequently overlooked symptoms. The late symptoms consist of pain (especially if there is pleural involvement), purulent expectoration (even masses of tissue may be coughed up), dyspnea, loss of weight and strength, clubbing of the fingers, extension and metastases (hoarseness due to recurrent laryngeal nerve paralysis is not uncommon) and profuse hemorrhages.

It is the matter of the hemoptysis which I wish to consider more fully. This sign quite frequently comes in for its share of prominence in the symptomatology of lung cancer, the only peculiarity being its small amount and repetition. Together with these repeated mild attacks of hemoptysis, there may be elastic fibers, leucine and tyrosine found in the sputum. Despite the fact that the tetrad of symptoms, namely, continuous thoracic pain, cough, dyspnea and hemoptysis, are considered to be of distinct diagnostic value, a recent statistical survey by S. A. Levinson,[2] of a large series of lung cancers which were confirmed at autopsy, less than 20 per cent had hemorrhage as a symptom, most of them being of small quantities.* I know from my own experiences and those of others that hemorrhage has been very characteristic, although some consider the presence of blood streaking not necessarily an indication of cancer. We are repeatedly called upon in cases to rule out a suspected area in the chest in the tuberculous as well as the nontuberculous when the cause of the hemorrhage is questioned.

A very common finding in lung cancer is the presence of large bronchiectatic cavities, the figures being anywhere from 30 to 50 per cent of the cases.[3] This is not unusual inasmuch as a bronchus, if occluded, signs of atelectasis of the part of the lung supplied by that bronchus may be found. A bronchiectatic cavity may result from secondary infection above the occluded area. Such cavities may

*In a series of 1386 autopsies over a five year period (1929-1934) from Michael Reese and Chicago Memorial Hospitals, Dr. Otto Saphir (personal communication) reports 19 carcinomas of the lung in which only 5 had hemoptysis, one of them dying from a huge hemorrhage. The histories were incomplete in 9 cases.

In a recent communication to the author, C. L. Jackson stated that hemoptysis has been a prominent symptom in at least two-thirds of their cases and that bronchoscopic study confirmed the diagnosis in about 75 per cent of the proven cases. He stated that he knew of a fatal hemorrhage following a bronchoscopy which was done in another clinic and also of another massive hemorrhage in a case which had been done several months previous to the fatal termination.

expose some of the larger vessels so that hemoptysis is said to occur in bronchiectasis in about 35 per cent of cases.[4]

In a discussion of the indications for bronchoscopy in tuberculosis, Clerf[5] states that only too often has hemoptysis led to errors in diagnosis, being so common in cancer of the bronchus and in bronchiectasis. He reviewed the case histories of a large number of patients with pulmonary suppuration and found that the occurrence of hemoptysis contributed more often to an erroneous diagnosis of tuberculosis than any other signs and symptoms. A majority of these patients had bronchiectasis diagnosed by bronchoscopy with the instillation of iodized oil. Clerf definitely states that hemoptysis does not contraindicate bronchoscopy unless the blood loss is great and continuous, and certainly obscure hemoptysis is an absolute indication for bronchoscopy.

It would seem that to most observers the picture is generally well known that neoplasms of the lung are accompanied by a raspberry jelly-like sputum. This likewise holds true for cancer of the bronchus. Intense fatal hemorrhages, however, are so rare that the literature contains but little reference to them. Maresch[6] records a series of 292 cases of carcinoma of the bronchus observed over a period of ten years. In this series ten fatal hemorrhages occurred. Rupture of vessels is not so very common following malignant growths of the lung as one would believe, knowing that metastases are ofttimes found to invade the pulmonary vein. The histories of repeated hemorrhages, even in large quantities, seldom have recorded a fatality. Although Maresch does not state so specifically, the deaths in his series were the result of spontaneous hemorrhages.

In a recent contribution, Reinhoff and Broyles[7] describe a patient whose hemoptysis was in such quantities that several cupfuls would come up from low down in the chest, after hearing a soft bubbling noise. The patient coughed up as much as four cupfuls a day. Bronchoscopically only a small polyp was seen in the left main bronchus. Attempts to remove this tumor were unavailing because it would result in considerable bleeding each time it would be tried. I have had similar experiences, one case still alive after five years, whose hemorrhages were severe but responded to fulguration and x-ray therapy. This case is of sufficient interest to place it on record.

REPORT OF CASES.

CASE 1.—M. J., a barber, born in Germany, age 50, was referred to the Research and Educational Hospitals of the University of Illinois in June, 1929, complaining of a persistent and productive cough for 3 years. The patient stated that

he was well until 1925 when he had dry pleurisy. In 1926 he had influenza. He was confined to the Municipal Tuberculosis Sanitarium from December, 1928, until February, 1929, where he was treated for bronchiectasis. His findings repeatedly showed atelectasis or fluid at the base of the right lung, posteriorly, but several aspirations over a period of 3 years gave no evidence of fluid. Hemoptysis had been constant but at the time he first was seen by us he had severe hemorrhages of several cupfuls of blood at a time. We bronchoscoped him and found pale papillary excrescences about 3 cm. down past the orifice of the right main bronchus, occupying two-thirds of the lumen. Bleeding was quite profuse. Tissue for bioposy was obtained which showed an adenocarcinoma. In October of 1929, I fulgurated the mass with surgical diathermy and the patient was given deep x-ray therapy. His hemorrhages ceased and he gained 65 pounds. In December, 1932, he returned to the clinic stating that he recently began to cough up blood again. Bronchoscopy was decided upon and during the process of cocainization of his larynx, a profuse hemorrhage occurred. A pinkish-grey, pearl-like mass obstructing the right main bronchus just below the orifice was seen to flap up and down on respiration. (Check-valve mechanism described by Jackson.) Blood was seen to come from the mass which was quite soft. Microscopic study again showed it to be adenocarcinoma. The patient was again placed on deep x-ray therapy. His hemoptysis ceased and he has been clinically well as far as his lungs are concerned. Other manifestations within the past year have aroused suspicions of metastatic extension to the bones of his pelvis, but nothing has ever been proven. He is now apparently in the best of health.

The experience of a sudden and fatal hemorrhage of any type is by no means a pleasant one. Bronchoscopy, it has been shown, is definitely indicated in cases of suspected malignancy and has gained such widespread favor that we may say it is universally used. Not recorded by title is a case of carcinoma of the lung reported by Vinson,[5] in which a fatal hemorrhage occurred after the introduction of the bronchoscope but before the lesion had been visualized or forceps had been introduced into the bronchus. There is no elaboration by Vinson as to the possible factors involved in the death. I also wish to report at this time the case of a fatal hemorrhage from the lung of a patient with bronchiogenic carcinoma following bronchoscopy.

Case 2.—J. K., was referred by the Cook County Relief to the dispensary on September 7, 1934. The patient was a white male, age 50, and a widower, born in the United States. Occupation, ice-man, but he had previously worked in a coal mine. Past history: measles and whooping cough; no history of contact with tuberculosis. The onset of his illness was insidious. He enjoyed good health until about April, 1934, when he had a cough with slight expectoration. There was no frank hemorrhage but occasionally he brought up a blood-streaked sputum. He complained of weakness and loss of weight (30 pounds within one year). His usual weight was 170 pounds, his present weight, 140 pounds. There was no fever, night sweats or pain in the chest; his appetite was good, the bowels regular; no dyspnea and no chills. The patient's nutrition was fair. He had a few decayed teeth, the heart was negative. There was an impaired resonance and the

breath sounds were diminished in the right upper lung. The urine was negative except for 100 mgm. of albumen. Sputum examinations were repeatedly negative for tuberculosis. The x-ray on September 19, 1934, showed a mass off the right hilus extending downwards, with a tentative diagnosis of bronchiogenic carcinoma of the right lung.

Bronchoscopy.—The patient was referred to the Municipal Tuberculosis Sanitarium for bronchoscopic examination on September 27, 1934. He was admitted at 9:45 A. M., and at 10:30 was given sodium amytal grs. iii. At 3:00 P. M. he was given morphine sulphate gr. 1/4. The pharynx and larynx were anaesthetized with cocaine topically. The bronchoscopic findings indicated a decided widening of the carina. The orifice of the right bronchus was almost stenotic, slit-like in a vertical direction. The irregularity was seen to come from the hilus area which produced a nipple-like protrusion into the lumen. The bronchus was stenotic 3 cm. beyond the orifice and no attempt was made to go further. A biopsy was taken from the region of the carina. The area felt very firm and no unusual bleeding followed. As the tube was being removed and just as the patient was about to sit up there was a gush of frothy, bright red blood. Re-introduction of the tube for suction and tamponage was impossible, the flow was so great. Heroic measures of resuscitation were unavailing and the patient succumbed in five minutes.

Postmortem Examination (Dr. Louis Parmacek).—"Thoracic Cavity: The diaphragm on the right is at the sixth rib; the left at the seventh rib. There is a focal fibrous adhesion from the right upper lobe to the chest wall, leading from a thick fibrous patch.

Lung Description: The lungs were removed in toto attached to the trachea by their respective bronchi. There was a palm-sized irregular, poorly demarcated glistening, white, fibrous plaque over the middle lobe and the adjacent portion of the upper lobe. Near its upper part, a few fibrous focal adhesions to the parietal pleura were seen. On attempting to inflate the lungs with air through a tube attached to the trachea, it was noted that the right lung failed to expand because air escaped from the upper right pulmonary vein. Only when this vein was clamped off did the right lung expand. The lungs were x-rayed and fixed in formalin. Dissection after fixation disclosed the following:

The trachea and stem bronchi contained much liquid and clotted blood. A 3x3 mm. defect 2 mm. deep was seen in the mucosa of the inferior medial wall of the right stem bronchus near the origin of the first hyparterial ramus to the middle lobe. Microscopic sections of the tissue from this site showed normal mucosa and fragments of cartilage with no evidence of malignancy. The middle lobe was firm, collapsed, and subcrepitant. Section through its hilar portion revealed a chestnut sized, irregular, infiltrating grayish, centrally softened mass surrounding and destroying the bronchus. A few similar satellite areas were seen. Several 2x1 cm. saccular, thin-walled, smooth lined cavities were also noted. The intermediate pulmonary tissue was inelastic. The right upper pulmonary vein, about 1 cm. from its point of penetration into the pericardial sac, presented in its superior dorsal wall a 4x3 mm. irregular tear communicating with the tumor mass above mentioned. Microscopic sections from this tissue and from the lymph glands showed squamous cell carcinoma without horn pearl formation.

Liver: Weight, 1330 gms.; smooth, deep brown, and firm. On the posterior surface of the left lobe a golf ball sized node protrudes. On cut section a central

liquified, 3 cm., gray, well-demarcated mass is seen. On the posterior aspect of the right lobe a firm 1½ cm. similar node is seen. A 3 cm. node is found near the one first mentioned. The liver substance is deep brown. The markings are indistinct. The sections taken from these areas likewise showed squamous cell carcinoma. The findings in the other organs revealed no related pathology or metastatic extension.

From the findings one may reconstruct the sequence of events as follows: The carcinomatous tumor mass was extending and softening. This extension involved the wall of the pulmonary vein, which became weakened. Under the added intravenous pressure from the strain of the presence of the bronchoscope the weakened venous wall ruptured into the softened tumor mass surrounding the bronchus. A massive fatal hemorrhage from the pulmonary vein into the bronchial tree was the result. The actual biopsy was in no way directly related to the hemorrhage, which may well have followed a severe coughing spell in the near future.

The emphasis upon hemoptysis as a symptom of lung cancer, together with the presentation of case reports from the literature of fatal hemorrhage, serves to illustrate the possibilities of bronchoscopic interference in these cases. My experience, coupled with the findings of the proliferative and ulcerative changes found on postmortem in these pulmonary lesions, causes me to believe that similar fatalities have occurred before without special attention being directed to this possibility. The mechanical effort of a bronchoscopic observation, performed under the best conditions, may be sufficient strain to provoke a hemorrhage from a vessel whose walls are near a malignant mass and perhaps even invaded by the neoplasm. There can be no question as to the advisability of the bronchoscopic procedure, but it is well to bear in mind the possibility of hemorrhage.

There is a tendency to minimize the importance of a fatal termination in such cases. My greatest difficulty with regard to this case has been in discussing it with colleagues. They immediately decide that the patient was better off, knowing of the tremendous lung involvement with liver metastases. This is beside the point. The fact is that an unpleasant experience is best avoided or at least anticipated. Had I not been fortunate in obtaining permission for the postmortem my thoughts would have dwelt more on the possible errors in technic rather than thinking of the fatality in terms of the existing pathology.

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XIV.

POSTOPERATIVE BILATERAL ABDUCTOR PARALYSIS.*

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BALTIMORE.

A great deal has been written, from both a clinical and an experimental point of view, on paralysis of the vocal cords. Paralysis of one cord is a rather common occurrence, usually resulting either from some pathologic lesion or an operative procedure. Indeed it has been noted that about 1 per cent of persons examined in routine preoperative procedure for conditions not related to the thyroid gland have unilateral paralysis of the vocal cord.[8] The causes of this distressing, but not dangerous, condition are legion.

In bilateral abductor paralysis there is a loss of function of each crico-arytenoideus posticus muscle which normally abduct the cords, open the glottis, and permit free passage of air into the lungs. It is a comparatively rare disease, more common in men than in women and almost foreign to children.[6] It may be either central or peripheral in origin. If a lesion is central in origin, only partial paralysis will result. Peripheral paralysis is probaby always complete in the case of a single laryngeal nerve just as it is with other nerves. The resultant laryngoscopic picture is easily explained by unopposed action of muscles supplied by other laryngeal nerves. True peripheral abductor paralysis occurs as the result of a lesion minute enough to involve only abductor fibers at their distribution in the larynx. The fact that there are four separate nerves supplying the larynx probably accounts for the confusion concerning paralytic lesions there.[7] Crile once said unilateral abductor paralysis is unfortunate, but bilateral abductor paralysis is a tragedy.[2] It is with this disease, as it follows operative procedure, that my paper deals.

The necessity of laryngeal examination before and after an operation, whether it be a thyroidectomy or some other operation on the neck in the region of the recurrent laryngeal nerves, cannot be too strongly urged. Not only is it a comfort to the surgeon to know the condition of the larynx in advance of and subsequent to the operation, but such knowledge also has a beneficial effect on the pa-

*Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Portland, Maine, January 4, 1935.

tient. Should paralysis of one cord be detected before the operation, the operative procedure may possibly be altered to prevent bilateral abductor paralysis. The ancient adage concerning the ounce of prevention and the pound of cure is more than applicable to this condition, as prevention is, of course, the ideal treatment.

The most common cause of this form of paralysis following operation is, I feel, the pull on the recurrent laryngeal nerve during the process of removing the thyroid gland. Injury from forceps, knife or sponging is not so frequent, though these possibilities should be kept in mind. Any direct trauma during operative procedure, or direct pressure by or involvement in a pathologic condition in the region where the nerves are located, is likely to produce this condition. The capsule and the nerve are in close proximity and the latter can easily be traumatized. This fact is borne out by the report of Fowler and Hanson,[2] who found in their examination of 200 cadavers that the posterior capsule of the thyroid gland left the gland at the postero-lateral portion of its lobe and passed directly backwards to the prevertebral fascia, thus leaving the posterior surface of the thyroid lobe uncovered and in direct contact with the nerve. It is the posterior margin of the thyroid, lying between the capsule and the nerve, that must be preserved and protected during the operation.[2] It must be remembered that this nerve, which is a branch of the vagus and supplies all of the intrinsic muscles of the larynx except the crico-thyroid muscle, is very sensitive and delicate, one of the most vulnerable in the body, and is incapable of withstanding trauma of any kind, as some of the other nerves can. Pressure or pull interferes with nerve conduction and paralysis quickly results. This may be of two kinds: partial and temporary or complete and permanent. During the past seven years I have seen eight cases of bilateral abductor paralysis resulting from operative measure, seven following thyroidectomy and one after removal of gland complicated by extensive infection and scar formation. Of these cases four were temporary and four showed no improvement after eighteen months or more. It is the interest and desire of the surgeon to assure himself and his patient that he has placed his case in the former class by means of measures taken to afford immediate relief. Paralysis resulting from scar formation usually appears late and may be relieved if it is not allowed to continue for too long a period of time. If the condition has existed for more than twelve months there is little chance of recovery. The crico-arytenoideus posticus muscle, after loss of function for a certain time, will atrophy and will not return to normal function. This is one reason why prompt relief is desirable.

It is the distressing symptoms produced by bilateral abductor paralysis which make it essential for the surgeon to devise a speedy measure of relief. I recall two cases which I saw a few years ago and diagnosed as bilateral abductor paralysis following thyroidectomy. The surgeon in each case was not satisfied with my opinion and declared the condition due to "hysteria." Alarming complications developed but little attention was paid to them. One of those cases proved to be temporary and the patient recovered. The other was less fortunate and died four days later, very suddenly, before aid could be administered.

What, then, are the indications pointing to the condition? We find definite hoarseness and some stridor. Later, hoarseness is less noticeable and stridor more marked. Especially is this noticeable with increased excitement or with exertion. A peculiar noise presents itself on inspiration, especially during sleep. Expiration is quite natural and unchanged. The patient dreads the attacks, and in time, unless relief is secured, becomes more or less of an invalid. Dyspnea increases as the voice, which may in time return almost to normal, improves. Direct laryngeal examination during cordal paralysis reveals cords and arytenoids in midline with only a narrow space between the cords. Their position is straight because the tensor function is not resisted. On inspiration the cords are drawn downward and inward, preventing the intake of air. On expiration the cords are forced upward by the outgoing current of air and there is a separation of the cord, more marked in the middle. There have been some cases reported in which the cords are in a cadaveric position which does not interfere with breathing but where the patient would still have difficulty in speaking. Personally I have not, in my limited experience, encountered any such cases.

The treatment begins with immediate tracheotomy, which alleviates the danger of asphyxia, removing the strain from the heart and affording time for study of the type of surgical procedure to be undertaken. If the case proves to be a temporary paralysis and the function of the cords returns, the tube can be removed and the opening closed without any ill effects. On the other hand, if sufficient time elapses to ensure that the condition is complete, one of the several types of operations should be selected and performed.

There are various kinds of surgical procedure, some successful and some which have failed in nearly every case. It was early found that cordectomy offered little or no relief. Jackson improved on this when he introduced ventriculocordectomy. This operation, while quite successful, produced a grave impairment of voice, which was,

of course, less serious than the imminent danger of asphyxia. The operation devised by MacKenty is not a difficult one, but it gives the necessary relief and at the same time preserves the voice. This operation aims to establish a small permanent opening in the trachea at a point just above where the trachea dips backward into the chest—the success of the procedure depends on securing a union between the skin and the mucous membrane, entirely free from scar tissue—the air passing through this small opening in the trachea adds to the air inspired through the natural breathway and conteracts the inspiratory pull on the cords. This pull is the chief factor in reducing the amount of inspired air. The stronger the pull the less air inspired. The opening also acts as a safety valve during the dangerous spasms of asphyxia so common in abductor paralysis. Should it be found, after this operation has been performed, that the normal function of the cords has returned, it is a simple matter to restore the larynx to its preoperative condition. This procedure may well be used as an intermediate step between tracheotomy and ventriculocordectomy which may be performed later. Indeed, where time and the condition of the patient permit, it may well supplant tracheotomy entirely.

Nerve anastomosis has been tried but with little success. Another type of operation is submucous resection of the cord. This has been tried by many with varying success. Hoover, of the Lahey Clinic, reported several operations of submucous resection which have been quite satisfactory. An ample airway is established and though normal voice is impaired a good whispering voice is assured. The object of this procedure is to widen the lumen of the larynx by the removal of soft tissue between the mucous membrane and the cartilage of the lateral wall, thus allowing the mucous membrane to be placed on the lateral wall, increasing the lumen and preventing the formation of granulations. A tracheotomy is the first step in this procedure, if one has not already been performed. An important point to be emphasized is that all tracheotomies should be done below the cartilages entering into the fundamental structure of the larynx, preferably below the second ring of the trachea. To make the opening higher than this is to invite a possible permanent dependence on a tube.

In summary, let me stress once more the importance of a laryngoscopic examination before and after thyroidectomy. Should the paralysis present itself following operation, treatment is indicated by the condition of the patient.

REPORT OF CASES.

CASE 1.—Mrs. D. I., aged 19, a housewife, was seen in consultation, complaining of nervousness, tremor and attacks of asphxia. Six months previous to

this a thyroidectomy was performed. Convalescence was slow. For two months she had a whispering voice, which improved but nervousness and asphyxia became more marked until the attacks were alarming. She was put on calcium therapy, advised to rest, and tracheotomy was performed. Indirect laryngeal examination showed both cords and arytenoids motionless and in midline. Her condition improved, except for difficulty in breathing without means of a tube. Cords have remained in midline with no evidence of recovery of paralysis after eighteen months.

CASE 2.—Miss C., a school teacher, following an operation for removal of glands and extensive infection, later developed dyspnea. Attacks of dyspnea became alarming and she was admitted to the hospital for observation. Indirect examination revealed a bilateral paralysis. There was no improvement after several months and under intratracheal anesthesia scar tissue was resected from region of recurrent nerve. Following this operation there was a gradual return of cords to normal movements.

CASE 3.—Mrs. D., aged 36, presented herself to bronchoscopic clinic for shortness of breath. Three months previous she was operated upon for thyroidectomy, from which, as she stated, she never completely recovered. At first she had hoarseness which cleared up, but had attacks of dyspnea and nervousness. Indirect examination showed paralysis of both cords. Following tracheotomy she was relieved of the distressing symptoms. After one month the cords were in midline. Further observation was not possible. This case had not been examined before or after operation until she came to the clinic.

CASE 4.—Miss D., aged 26, was operated upon for thyroidectomy. Immediately following there was hoarseness with some dyspnea. Laryngeal examination showed complete paralysis of the left cord. A few days later there was paralysis of the right cord, with stridor. The voice improved and likewise the condition. After a few weeks paralysis had disappeared.

CASE 5.—Mrs. B., a housewife, was being operated upon by Dr. C. W. Maxson for substernal thyroid. Suddenly she became cyanosed and was unable to inspire. Immediate direct examination revealed both cords in midline. A hard rubber French catheter was inserted between the cords into the trachea. Her condition immediately improved and they were able to continue with the operation. The catheter was removed late the same day. There was some dyspnea for a few days, but paralysis gradually disappeared and she made a good recovery. Comment: This condition was due to bilateral paralysis, due to pulling on nerve in delivering gland. The insertion of catheter permitted operation to be finished and possibly saved the patient's life.

CASE 6.—Mrs. K., aged 41, housewife, was seen in consultation on account of stridor following thyroidectomy three months previous. Following removal of the thyroid she was hoarse for six weeks. When this improved the stridor increased in severity. Indirect examination showed both cords in midline. There was a very narrow space in the glottis between cords, with no abductor motion. The obstruction during the attacks was so severe that there was retraction of the sternal notch and intercostal space. There was no interference with expiration and the voice was fairly good. Immediate tracheotomy was advised, but was not permitted by the surgeon, as he felt this was largely a nervous condition. Four days later she died suddenly before aid could be administered. Comment: Tracheotomy would have saved the patient's life. Hysteria does not produce objective or subjective symptoms such as this patient presented.

CASE 7.—Mrs. M. R., aged 56, a housewife, was referred to a clinic for laryngeal examination. She had had a thyroidectomy three years previous, following which she had nervousness and difficulty in breathing, especially on exertion or excitement. Examination showed she was wearing tracheotomy tube. She appeared well nourished. Basal metabolism was within normal limits. There was hypertension and cardiovascular disease of long standing. The patient was seen by Dr. Chevalier Jackson, who made the following suggestions: That she wear a valve canula which would allow comfortable breathing and talking; that a partial cork used during the daytime could be removed at night for comfortable breathing; that the outer canula be removed daily and cleaned and that the inner canula be removed and cleaned as often as necessary; that care be taken to see that the canula fits perfectly, so as not to hinder free passage of air. Both cords were in midline and there was only a narrow space present between the ends on expiration. This was due only to the force of expired air. Because of the age of the patient and the duration of the condition she was advised to use a tracheotomy tube, since she was comfortable and able to talk without difficulty.

CASE 8.—Mrs. W., aged 42, was admitted to Mercy Hospital November 21, 1928, for enlarged colloid thyroid. Laryngeal examination showed both cords normal. Following thyroidectomy, under gas anesthesia, there was a short period of hoarseness and rapid pulse. This improved and dyspnea became more marked. Laryngeal examination revealed both cords in midline and no abductor movement. The patient was seen again on January 6, 1929, at which time the paralysis had completely cleared up.

MEDICAL ARTS BUILDING.

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XV.

ROENTGEN DEMONSTRATION OF CYSTS IN THE UPPER AIR PASSAGES.*

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Cysts in the upper air passages are relatively rare. When present they are frequently demonstrable on the roentgenogram. This is particularly so when they encroach upon the air spaces of the larynx and pharynx. During the year 1933, while making routine roentgen studies of the neck for laryngeal tuberculosis,[14] we found three cases with laryngeal cysts. These cysts were accidental findings. In addition, we present two other cases: one, a large sublingual cyst (ranula), about the size of a hen's egg, which displaced the tongue; the other, a thyroglossal cyst which was recognized by indirect findings.

LITERATURE.

A review of the literature reveals very few instances in which roentgen studies were utilized to demonstrate the presence of these cystic masses within the upper air passages. Donilan,[4] in 1912, made an unsuccessful attempt to demonstrate an epiglottic cyst by roentgen examination. In 1933, Myerson[11] reported three cases of laryngeal cysts and illustrated these cases with roentgenograms.

When compared with other lesions of the larynx, cystic tumors are rare. They are not as rare, however, as one is led to believe by the relatively few references in American textbooks on otorhinolaryngology. Several textbooks do not mention the subject at all except to enumerate the types of benign tumors found in the upper air passages. Myerson states that cysts are found less frequently than the literature of twenty to thirty years ago would indicate, there being no doubt in his mind that polypi of various types were included at that time. In 1871, MacKenzie[8] collected one hundred cases of laryngeal tumors and found cysts in only two cases. In Beschorner's[1]

*From the Roentgen-ray Department, Sea View Hospital, New York City.

collection (1877) of 693 polypoid tumors of the upper air passages, forty-five, or slightly more than 6.5 per cent, were cystic tumors.

In 1881 Moure[10] reported 117 cases (quoted by New[11]) of laryngeal cysts, of which eighty-two were collected from the literature and thirty-five were his own. The anatomic distribution of these cysts was as follows:

Epiglottis	50
Vocal cords	45
Ventricle	8
Arytenoid cartilages	4
Aryepiglottic folds	3
Cartilago Santorini	1
Ventricular bands	2
Location not indicated	4

Jackson and Coates[7] list thirty-five cases of laryngeal cysts, twenty-three being epiglottic in origin. Laryngeal cysts, in the order of their frequence, as listed by Coakley,[3] are as follows:

Ventricular bands
Aryepiglottic folds
Epiglottis
Ventricle of larynx
Rarely from vocal cords

Gleason[5] lists the following benign *tumors* of the larynx in the order of their frequency:

Papilloma
Fibroma
Angioma
Myxoma
Cyst or cystomas

Myerson quotes Mishkin,[9] who reported seventy-six cases of epiglottic cysts; forty-nine were on the lingual aspect, ten on the laryngeal aspect and seventeen on the rim.

H. M. Taylor,[13] in a review of the literature up to 1923, commented that the majority of laryngeal cysts were intralaryngeal in location; and that less than 50 per cent were situated on the epiglottis. Most epiglottic cysts were located on the lingual surface, the frequency being six on the lingual surface to one on the laryngeal surface. He also quoted Purseagle: that 40 per cent of all tumors of the epiglottis reported and collected up to 1905 were cysts. In this series he believes degenerated polypi were included.

PATHOLOGY.

Boyd[2] divides cysts in general into four groups:

A. Retention cysts
B. Cysts of new formation.
C. Parasitic cysts
D. Cysts of developmental origin

The retention cyst is the most common and is usually small. It may vary in size from a small pea to the size of a hen's egg (reported by Hamilton[6] in 1899). The retention cysts may be located in any part of the larynx. The common sites are on the lingual surface of the epiglottis, and in the ventricle, where mucous glands are numerous. Cysts of new formation are due to extravasation of blood or lymph, found usually about the vocal cords. The embryonic types or cysts of developmental origin are found usually on the aryepiglottic folds and on the lateral laryngeal wall.

SYMPTOMATOLOGY AND DIAGNOSIS.

The symptoms produced by these cysts vary greatly, depending upon their size and location, being either entirely absent or manifested by one or more of the following: a desire to clear the throat, a change in voice, hoarseness, cough, cyanosis, dysphagia, stridulous breathing or dyspnea. Three of our cases presented no symptoms and were accidental findings.

The diagnosis may be made by direct, mirror or digital examination. The roentgen examination of the soft tissues of the upper air passages has not been sufficiently stressed. Roentgenologically, one is able to demonstrate the presence of a mass, if it has attained a given size and encroaches upon the upper air passages, or is large enough to produce symptoms by pressure or displacement of other structures. The air serves as a contrast medium. In many instances the size, shape, location and site of attachment of the cyst are better demonstrated by the roentgen examination than by any other method. The roentgen study is the only means of obtaining a sagittal view of the structures in the neck. Most of the epiglottic cysts occur on the lingual surface. When small, and situated deep in the vallecula, they are not easily recognized.

REPORTS OF CASES.

CASE 1.—J. M. Male, white, age 49. Admitted to Sea View Hospital with symptoms of pulmonary tuberculosis. Physical and roentgen examination of the chest showed the presence of a tuberculous pulmonary lesion of a caseous pneumonic type involving the right upper lobe with a diffuse acinous exudative spread

throughout both lungs. Oral examination showed a large cystic mass arising from the floor of the mouth displacing the tongue upward and backward. The patien. stated that this mass had been present since childhood and varied in size from time to time. Fig. 2.

CASE 2.—J. B., male, white, age 32. Admitted to Sea View Hospital with symptoms of pulmonary and laryngeal tuberculosis. Physical and roentgen examination of the chest revealed a bilateral caseous pneumonic type tuberculosis involving the upper lobes. Roentgen examination of the larynx revealed the presence of a sharply circumscribed soft tissue mass measuring about one-half inch in diameter in the region of the upper half of the epiglottis. The arytenoid eminences were enlarged. The ventricle was irregular and deformed by an inflammatory process. Fig. 3. Mirror examination revealed the presence of a cystic tumor mass attached to the right side of the epiglottis. The arytenoid eminences were injected and infiltrated. There was a hyperplastic and ulcerative lesion involving the true cords.

CASE 3.—L. K., male, white, age 40. Admitted to Sea View Hospital with a diagnosis of pulmonary tuberculosis. Clinical and roentgen examination of the chest revealed a chronic proliferative type tuberculosis involving the right apex. Roentgen examination of the neck revealed the presence of a small mass deep in the vallecula (Fig. 4). Laryngeal examination revealed a cyst the size of a small grape on the lingual surface of the epiglottis.

CASE 4.—V. A., Female, white, age 18. Admitted to Sea View Hospital with a diagnosis of pulmonary tuberculosis. Clinical and roentgen examination revealed a caseous pneumonic type tuberculosis involving the right upper lobe. Routine roentgen examination of the neck showed the presence of a small circular sharply circumscribed mass, the size of a pea, arising from the aryepiglottic fold near the tip of the epiglottis (Fig. 5). Mirror examination revealed a cyst the size of a pea on the left aryepiglottic fold. The remainder of the larynx showed no abnormal findings.

CASE 5.—C. S., male, age 54. For one year complained of cough, irritated throat, and mass in front of neck which varied in size. At times it produced a visible mass, approximately one inch in diameter. Other times it would disappear entirely. Roentgen examination revealed an upward displacement of the anterior portion of the hyoid bone, and a backward displacement of the lower portion of the epiglottis, by a mass measuring about 2 cm. in diameter (Fig. 6). A thyroglossal cyst was found and removed (by Dr. Louis Franklin, Jersey City).

SUMMARY.

Five cases with cysts in the upper air passages are demonstrated roentgenologically: a ranula, a thyroglossal cyst and three laryngeal cysts. The laryngeal cysts were accidental findings and presented no clinical symptoms.

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Fig. 1. *Air Spaces:* 1. Pharynx. 2. Pyriform sinuses. 3. Epiglottic vallecula. 4. Laryngeal vestibule. 5. Laryngeal ventricle. 6. Trachea. 7. Air in cervical esophagus. 8. Apex of lung.

Cartilages: 9. Epiglottis. 10. Cartilago triticae. 11. Thyroid cartilage. 12. Arytenoid cartilage. 13. Cricoid cartilage.

Soft Tissues: 14. Uvula. 15. Tongue. 16. Posterior pharyngeal wall. 17. Aryepiglottic fold. 18. Fat pad between epiglottis and thyrohyoid membrane. 19. Pharyngopalatine fold. 20. False cord. 21. True cord. 22. Arytenoid prominences. 23. Postcricoid area. 24. Cricothyroid membrane. 25. Retro-tracheo-esophageal area. 26. Sternomastoid muscle. 27. Thyroid gland.

Bones: 28. Hard palate. 29. Hyoid bone. 30. Atlas. 31. Axis. 32, Mandible. 33. Mandibular condyle. 34. Clavicle. 35. Styloid process.

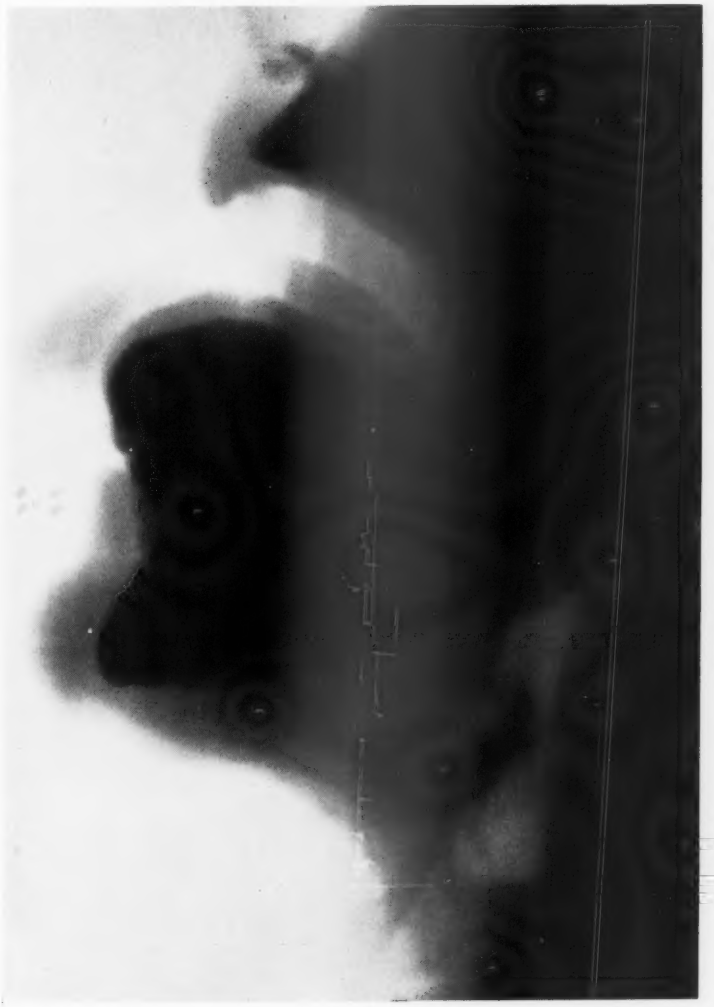

Fig. 2. Large cystic mass displacing tongue.

Fig. 3. Cyst in region of upper portion of epiglottis.

Fig. 4. Cyst in vallecula against lingual surface of epiglottis.

Fig. 5. Small cyst on aryepiglottic fold near tip of epiglottis (immediately below hyoid bone).

Fig. 6. Upward displacement of anterior portion of hyoid bone by a thyroglossal cyst.

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XVI.

THE TREATMENT OF RHEUMATISM.*

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SAN FRANCISCO.

The gradually developing conception that rheumatism is a systemic disease due primarily to some error in our fundamental life that encourages infection has, it seems to me, tended to discourage the efforts of our surgeons to remove the foci of infection. This is so contrary to my feelings in the matter that I determined to review the subject from the standpoint of the work done in the otolaryngologic department of the Southern Pacific Hospital, where some 3000 tonsillectomies have been done since 1927.

It is well known that rheumatism is a general term covering a multitude of pathologic lesions, each worker classifying the cases according to his best knowledge of the subject, but the classifications themselves give us a fine lead toward treatment and a hope for a better knowledge of the subject in the future.

Burbank and Hadjopoulos,[1] after a prolonged serologic study, made an illuminating classification of arthritis into

(A) Arthritis reacting to hemolytic streptococci and belonging to the isoatrophic class (periarticular).

(B) Arthritis reacting to hemolytic streptococci but of different fixing properties. Type anisoatrophic or deformans.

(C) Arthritis reacting to streptococcus viridans type and belonging to the osteo-arthritis or productive form.

Although these soon became mixed types, these workers leaned strongly to the opinion that the disease was definitely of bacterial origin and specific in character; that is, that each organism produced primarily a definite type of lesion.

Hench, 1930,[2] classifies the disease into

1. Infectious.
2. Traumatic.

*Read before the Western Section meeting of the American Laryngological, Rhinological and Otological Society, Inc., January 19, 1935, Los Angeles, Cal.

3. Senescent.
4. Chemical, i. e., gouty.

This classification would seem to be ideal for treatment and seemingly would enlarge the field over that of Burbank.

Again, Cecil[3] calls attention to a more recent classification:

1. Infectious (rheumatic fever; rheumatoid arthritis; arthritis caused by specific organism).
2. Degenerative (osteo-arthritis or hypertrophic).
3. Allergic.
4. Traumatic.
5. Metabolic (gout, scurvy; rickets).
6. Neurogenic arthropathy (syringomyelia, Charcots posthemiplegic, etc.).

The American Rheumatic Commission[4] has classified the disease into

1. Atrophic, including conditions known as proliferative, rheumatoid, ankylosing, metastatic and Stills disease.
2. Hypertrophic arthritis, including degenerative osteo-arthritis; menopausal senescent, nonankylosing, von Bechterew syndrome and the Marie Stumpel syndrome.

They threw together the specific and such as those of gonorrhea, syphilis, gout, trauma, rheumatic fever, tuberculosis, tabes, hemophilia, purpura, etc.

The Ely and Fisher classification is:

	Ely	Type I	Type II
X-ray appearance	Goldthwait	Atrophic	Hypertrophic
Histogenetic	Nichols and Richardson	Proliferative	Degenerative
Etiologic		Bacterial Lues Tb. Neisser Staph. Strep. Etc.	Protozoal (?)

Rheumatoid arthritis is Type 1 in histogenesis but no bacteriologic etiology known.

Marie Stumpel type is Type II in its histogenesis but has some of the late characteristics of Type I.

No subject can be approached with any great satisfaction that is accompanied by as great confusion in the nomenclature as is this, and still there is a glimmer of light through it all when one realizes that the individuals who have made the most intensive studies of the subject lean to the opinion that bateria are at least a potent cause of the symptoms complained of and of the pathologic lesions present. That it may be a systemic disease is true, or that the general condition of the patient may determine the character of the local lesion or the rapidity of its attack, etc., but this is true of any other disease and not limited to rheumatism. The multiplicity of lesions found in tuberculosis and the varying intensity of attack is no more profound than is that of rheumatism, yet there is no question about the cause of the disease. The picture in the majority of the rheumatic cases is that of a bacterial invasion modified by the character of the host and the virility of the organism.

If there exists a constitutional derangement, such as lack of iodin, calcium, magnesium, sulphur or a too pronounced carbohydrate or protein diet or a dilated or elongated colon or a defective posture producing a trauma, as is claimed by many, it still remains that the ultimate lesion is caused by a bacterium and that this bacterium must be eliminated as soon as possible in order to give the host a chance to gain its equilibrium.

It is true that the body may become immune to the presence of bacteria, and if the body is provided with the necessary help the rheumatic tendency may be shunted, but there is no reason why that should be the main effort on the part of the physician in attendance if he once has a clear grasp of the ravages which may be meted out to the patient if the infection is not properly cared for. If we can show that there is a direct relation between a focus of infection and the diseased joint, in some cases it is more than proper to at least remove all foci as a preliminary to any other treatment, even though there is a doubt in the physician's mind as to the real origin of the disease.

To the 3000 tonsillectomy patients at the Southern Pacific Hospital questionnaires were sent asking if the operation was done on account of rheumatism, and, if so, what was the result. There were 1257 replies (many of the men were no longer in our employ and did not get the paper). Of these, 819 were done for reasons other than for rheumatism. Of the remainder, 300 claimed they were entirely free of rheumatism, 100 were improved to various degrees and 38 claimed that there was no improvement.

Naturally the question arises as to how many of all these actually had what is called rheumatism; that cannot be ascertained, but it must be assumed that a large proportion knew what they were coming to the hospital for. Most of these cases were among people of from thirty to fifty years of age, males, and the majority from an intelligent class. On reviewing the histories of those claiming no improvement, I found that the larger proportion had had their trouble for many years and showed deformities by the x-rays. The histories of some of this group did not mention any rheumatic pains but stated they entered hospital for tonsillectomy on account of repeated sore throats. On questioning the various members of the staff, I found it was a unanimous opinion that the removal of the foci had in the majority of cases been the prime factor in the cure of the cases.

My own observation, and I have questioned many minutely, is that there occurred immediately on removal of the tonsils an amelioration of pain or swelling or of both; this often occurred before the patient reached his bed, and that certainly is what one could expect if the pain is due to a toxin circulating in the blood stream specific for a certain joint. I have had acute cases come in on crutches and leave the operating room without them. Many of these cases did not get well until infected teeth were removed or the prostate gland was treated or a nasal sinus cleared out, but this only is confirmation of the opinion that the immediate cause of the joint lesion was an infection.

One of my cases was a laryngologist who was gradually becoming helpless. He had, over a period of years, hunted everywhere for a focus. He had never had sore throats and his tonsils were so small and innocent looking that both he and I doubted their being the origin of his trouble. At operation I opened an abscess as large as one's thumb nail and at once all arthritic symptoms disappeared so that within six months there was normal movement of the spine and limbs. This is not an isolated case, but well illustrates the point I wish to make, that there is some definite relationship between a focus of infection and a pathologic lesion in the joint.

I purposely made no attempt to separate the cases with the atrophic from the hypertrophic forms, preferring to let the patient himself say yes or no as to the presence of what is popularly known as rheumatism, thus getting a cross section of the results that might be obtained through removal of focal infections. This may not be scientific but it proved to be highly illuminating, for it must be granted that in the class of cases operated upon the disease that is

most common is the osteo-arthritis and not rheumatoid arthritis, and the staff of the hospital agrees that the majority of the cases seen were of the former type.

Conceding that the rôle of a bacterium is important, at least in the production and therefore the treatment of arthritis, it is just as necessary to go further and inquire into what causes the infection to become manifest and into the possibility of controlling that factor.

On questioning physicians who have practiced in the Philippines and in Japan, I find that there is very little rheumatism in these places. It has been suggested that in neither place are proteins consumed in anywhere near as large quantities as in America or Europe and that this large protein consumption in the latter countries produces a fermentation in the bowel which in turn causes a swollen mucous membrane and glandular system with its subsequent train of infection with its sequences. Keeping this in mind, one can well understand the desire of one group of men to cure by removing the colon or by high enemas.

Another suggestion has been made that it is the dominance of iodin in the Eastern diet that decreases the incidence of rheumatism. Again, in these countries, there was formerly a preponderance of foods untampered with by manufacturing processes: whole rice, mushrooms, greens, fish, fruits, instead of prepared grains from which all the important and necessary qualities have been removed, canned milk, vegetables, preserves, sugared goods, frozen meats, that have hung for a month, etc.

When we think of what happens to a kernel of wheat as it is prepared for white flour we wonder that there is any nutrient value left. What we are handed out is pure starch bleached by H_2SO_4 in order that it will remain dry for shipment. Vitamin, oil, roughage, etc., have all been removed. If we could grind the raw kernel as in former times a great many of our modern diseases might take on a different appearance, and this applies to a large list of prepared foods.

The result of feeding prepared foods to chickens is well illustrated by the experiments carried out by one of the patrons of Park and Pollard, "who divided a hatch of 1000 chickens, placing 500 in one brooder and the remaining 500 in another brooder in adjoining pens, all conditions as identical as possible to make them, with the exception of the feed, the first pen being a commercial ration overmineralized with inorganic materials. We say 'overmineralized' for the reason that a later test of the feed developed a 12 per cent ash content in the ration. This group of chickens was very nervous,

lacked one or two points of pigmentation in the feet and beaks, as compared with the second pen, were as flighty as young quail, fearful of shadows passing across the pen and could not be caught except by panicking them into a corner and then seizing a handful of them. Whereas the second pen, fed upon a simple grain ration containing liberal quantities of cornmeal and our seaweed product, were more tame than the customary groups of Leghorn chickens; in fact, the attendant—a woman—at any time she bent over in the pen, the youngsters were gathered around and picking at her hands and talking to her as contented chickens always do. The pigmentation in the beak and legs was as previously noted, two shades or more deeper than the other lot. They carried their heads lower naturally, and were very easily handled; broke to roost very easily, whereas the other pen would not content themselves on roosts until two or three weeks later."

If this occurs in chickens, the same or similar reactions undoubtedly occur in the human, and the fine studies now being made on the various constituents of our food products, such as the iodin in the milk, the mineral content in vegetables from different parts of the country and numerous other investigations into the food intake are going to have a profound influence in our conceptions concerning rheumatism.

It is still possible that, as in beriberi, we may turn to the East for a solution of the problem as to how to attack the fundamental cause of our infections, but until that time we shall have our enthusiasts using calcium ortho-iodoxy-benzoate, colloidal sulphur, gold, HCL., bicarbonate of soda, salicylic acid, vaccines, vitamins, internal gland secretions, soluble antigen and x-ray, but in my experience the thing that will save the greatest time for the present still remains the thorough elimination of the foci of infection.

490 Post St.

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XVII.

TONSILS AS FOCI OF SYSTEMIC INFECTION.*

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The subject to be discussed is: "Tonsils as Foci of Systemic Infection." In order that the words "tonsils," "foci" and "systemic infection" may convey the same meaning to each of us they must be defined.

1. *Tonsils,* the pharyngeal and the lingual. These represent specialized lymphoid tissue. However, any tissue of the same composition, even if not unified structurally with separate vascular nerve supply, must be considered as important in its relationship to direct infection and the transmission of infection, either through lymphatic channels or by allergic reaction of the body. In other words, the nasopharynx contains a great deal of tissue which is very similar in composition. Some is called lymphoid tissue, and the more specialized tissue, tonsil. Therefore, the word "tonsils" means lymphoid tissue, other than nodes, above the seventh cervical vertebra.

2. *Foci.* A focus of infection is understood as a locus containing bacteria or inflammatory tissue, with or without the constant presence of bacteria ascertainable.

3. *Systemic Infection.* First, the entire body may be involved. Second, various systems of the body, such as the cardiocirculatory, genito-urinary, bones or joints, etc., may be specifically or singly involved.

4. The problem restated then is: Can the lymphoid tissue present above the seventh cervical vertebra be a focus or transmitting agent for the production of infection or disease in one or more systems composing the body structure? Is there immediate, delayed or constant response to acute, subacute or chronic inflammatory change in the lymphoid tissue?

It is difficult to prove that such infection is the activator or producer of the systemic change in the body, but, if proved, extir-

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pation of this source of infection logically follows and, with extirpation, halting the progress or advance of any systemic disorder which has depended for existence on the intermittent or continuous manufacture of toxic irritating factors, either by direct transmission or by the production of an allergic reaction in other tissues of the body.

In my experience there are four types of tonsillar and nasopharyngeal reactions indicating infection which may affect systemic disorders.

1. There is the acutely infected tonsil, with and without abscess formation. This is easily recognized.

2. There is the red, hypertrophied chronic tonsil with and without symptoms. This also is easily recognized and is obviously infected. However, the individual possessing such tonsils is often completely free from any subjective symptoms and is unaware of any relationship, subjective or objective, between his throat and systemic disease when present. There is constant absorption from such infected areas, though the toxic or bacterial activity may be of a mild, low grade type.

3. The third reaction is not easily recognized, and plays a most important part in connection with systemic response to its toxic irritants. In this tonsil the anterior pillar, particularly the portion overlying the upper half, is a purplish red color, as are the edges of the soft palate, and at times the uvula. If this throat is observed closely from time to time, the appearance will be seen to change from the dull, purplish red to a much more brilliant inflammatory shade, but with the underlying purplish tinge remaining and becoming more pronounced. This is the type of infection in which a sore throat is rarely mentioned unless to it is added exogenous infection. Exogenous infection means an infection entering the body from without; endogenous infection, one which is carried in the body. The difference between exogenous and endogenous infection is in the duration of the changed appearance of the throat. In endogenous reaction, with or without systemic response, the tonsillar picture subsides quickly and returns to its former chronic appearance in a day or so; the systemic reaction, if recognized, is also of short duration. Exogenous reaction is actually a subacute tonsillitis or an acute exogenous infection superimposed upon a previously chronically infected or functionally destroyed tonsil.

This type of tonsil may have retrotonsillar adhesions, abscesses or, what is more likely, under the congested discolored anterior pillar

and soft palate, a single large abscessed region, or numerous small pockets, which may or may not open to the surface. This is concealed in a cursory view of the throat, but if the anterior pillar and soft palate are separated, will be seen. This type of tonsil is of particular importance because it may be a transmitter of direct infection or may produce allergic reaction on the part of the body without subjective sore throat. (This throat differs from the well known "smoker's throat," which shows obvious pharyngeal, rather than tonsillar, irritation.) My attention was first directed to this type of nasopharyngeal infection in 1918, when I was called to see a woman of sixty, complaining of brilliant red, swollen, painful, distal phalangeal joints, with moderate fever but no sore throat. These joints had heretofore showed degenerative changes but without symptoms. Obviously some additional factor had produced this change. Though subjective throat symptoms were denied, local examination showed two small, brilliant red spots in the tonsillar fossæ (the tonsils had been removed years previously). Culture from these spots showed a growth of streptococcus only. Within ten days the throat had resumed a perfectly normal appearance to my then inexperienced eyes. The small red areas were no longer visible. Coincidentally inflammation in the joints had also subsided. Later the same sequence of events was noted. Consultation with various otolaryngologists followed. Each reported the throat as negative for infection, or as a source of whatever had activated the degenerative joint changes to acute reaction. Eventually a consultant saw the patient when the throat was again at the height of a flare-up, again without local symptoms but with the same joint response. The same red tabs were observed on top of a dusky purplish area. During the next period of quiescence these tonsillar remnants were removed, and for the remaining ten years of her life the patient experienced no further joint flare-up.

Similar clinical observations were made in the ensuing years. In 1919, a series of patients with and without tonsils, suffering from various chronic diseases, was studied. The group showed from time to time flare-up of systemic disorder, with moderate fever of a few days' duration; and if arthritics, definite joint reaction; but no subjective throat symptoms. In each instance, examination of the throat showed this same marked discoloration of the membrane above the tonsil—that is, the anterior pillar, soft palate and uvula. According to the short or prolonged duration of the flare-up, the reactions were considered as of endogenous or exogenous origin. Fatigue, loss of sleep, emotional upset, etc., were some of the factors which produced systemic body reaction to this latent tonsillar infection. During the

period of quiescence these throats had the appearance of what I term chronic tonsils but which were not sufficiently obvious to interest the average otolaryngologist until he happened to see the same throat during its active stage. In this group of patients the tonsils had been considered and dismissed as playing a part in the production of recurrent joint flare-ups; but after repeated observation and the gradual recognition of this particular nasopharyngeal reaction it was agreed that the tonsils did play a part in the systemic disorder. They were removed during periods of joint and throat inactivity with gratifying results in a certain proportion of the patients. This third type of tonsillar reaction will be recognized only if the patient is watched day by day for some weeks, and sometimes for months. It will then become evident that this discoloration in the tissue subsides, disappears and reappears; but at all times there remains the underlying picture of chronic infection. Without repeated examination it often will be overlooked, ignored or misinterpreted, though it is an actual factor in the production or maintenance of chronic systemic disease.

4. The fourth type of infectious lymphoid tissue in the throat is the nasopharyngeal cavity itself, which is very often a greater source of infection than is realized. If it is not examined repeatedly as in the third type of throat, it may be considered as normal or of no importance. It is due to this type of tissue that certain tonsillectomized patients continue to have their former complaints of sore throat, colds, etc., with the same systemic reaction. Many of these individuals come for treatment of a severe head or chest cold, denying any sore throat in connection with the infection. More careful questioning may reveal a mild scrapy throat of such short duration, and so immediately followed by acute involvement above or below, that it had been forgotten. The nasopharynx, however, had certainly been the original host to the invading bacteria. This lymphoid tissue may well be considered as small atypical tonsils, subject to exactly the same infectious changes as the tonsils and probably responsible for much the same systemic reaction. Tonsils when infected may be compared to lobar pneumonia: nasopharyngeal lymphoid tissue other than the tonsiles, when infected to bronchopneumonia, or many little pneumonias. Cultures from the nasopharynx during disease or infection show staphylococcus more frequently than they do any other type of bacteria, so that it has become almost axiomatic that nasopharyngitis in chronic disease is treated as a definite staphylococcal infection. On the other hand, true tonsillar infection cultures show mixed bacteria or more frequently streptococci. Cultures taken at different stages of an acute tonsillitis may show a complete cycle

of the streptococcal bacteria beginning with a nonhemolytic streptococcus, and winding up again with the nonhemolytic. Whether the staphylococcus is a pleomorphologic bacterium has not been proved satisfactorily to date. The direct effect of the nasopharyngeal infection, most frequently staphylococcic, in chronic disease has not been given sufficient consideration.

Shortly before leaving Boston I was very much pleased to find that twenty or thirty years ago Dr. Mosher had from clinical observation and some culturing at that time reached, or suggested, the same point of view that I am presenting here regarding the potential or probable staphylococci pleomorphology.

Recognition of infected nasopharyngeal lymphoid tissue must be followed by recognition of its influence upon the rest of the body. Clinical observations and years of practical experience have proved to the satisfaction of most clinicians what science has failed to prove as yet—i. e., that infection in the nasopharyngal structures has direct bearing on changes in the respiratory tract, the gastro-intestinal tract, the genito-urinary tract, the skeletal structure and the homostasis of the body. Whether the first attack of tonsillitis ruins the functional activity of that tonsil, with or without retention of infection, is in a way beside the point, because other lymphoid tissue in the throat may be the transmitter of direct infection or the sensitizing agent for allergic reaction in other body systems.

Infection directly transmitted follows the lymphatic course, forming depots in the chest with hilus gland involvement, which, in turn, if inadequate in their function, produce local or diffuse distribution of infectious material. At any stage a few, or many, toxic elements or bacteria may enter the blood stream producing a definite bacteremia of minor or major degree. Also at times a localized abscess may be formed which, without proof of bacteria, may serve as a source of allergic response on the part of the body. Such response may be in the form of well recognized skin reaction—urticaria or hives—angioneurotic edema, hay fever, or possibly joint reaction of a type which may be termed hay fever in a joint. Allergic symptoms manifest themselves in two ways: First, spasm of smooth or involuntary muscles; second, vascular disturbance, probably increased capillary permeability.[1]

It is imperative, therefore, that intensive individual study of each patient be made; that the otolaryngologist have at his command the assistance of the laboratory, roentgenologist and clinician in order that the optimal time for operative interference be chosen. Unsatis-

factory results of operation, though they may be in the minority, have lessened unfortunately general interest in infection above the seventh cervical vertebra as a causative agent or transmitter of infection affecting the rest of the body. My own experience has proved to me that infection in the nasopharyngeal tissues has a definite effect on systemic disease; that in some instances it should be removed; in others it should not; that there is a time in each patient's course when operation will prove of definite benefit; that there is also a time when it will prove disastrous. The mere recognition of nasopharyngeal infection and the fact that it is definitely an extra load which has precipitated its host into some other systemic infection; caused a recurrence of a previously quiescent disorder, or pushed the patient over the borderline into a downhill course, is not of itself sufficient to warrant operative interference. Intelligent and successful choice of the time for operation depends upon study of each individual patient and should include:

1. A history that is complete in every possible detail.

2. A complete physical examination as well as a local examination.

3. Examination of the blood—the red cells, the white cells, hemoglobin, smear, sedimentation index. This will rule out any actual disease. Removal of infected areas during the height of activity of any systemic disease, especially during the acute inflammatory condition of joints in arthritis, is more likely to be followed by disaster than triumph. A possible exception to this is in the rheumatic fever syndrome. Here, even during the beginning or height of the acute condition tonsils are often better out than in. In time this may be proved also true for scarlet fever.

4. A seven-foot plate of the chest to recognize arteriosclerotic or hilus changes, unrecognized rheumatic heart, tuberculosis, bronchiectasis, so-called chronic bronchitis, or emphysema. Any of these findings indicate another reason for treating tonsillectomy as a major operation, "not to be entered into lightly or ill advisedly."

5. Examination of urine is indicated obviously for the presence of nephritis, pyelitis, hematuria, etc. However, it is so well recognized a fact that it is frequently forgotten, that a kidney may be damaged and still produce apparently normal single specimens of urine. Therefore dilution and concentration tests, phthalein output, etc., are as important pre- and post-operatively in tonsillectomy as in any other major operation. These tests indicate the actual physiologic

function of the kidney. Excellent detailed descriptions are given in Fishberg's recent book on hypertension.[2]

6. A clinical observation which should be given serious consideration is marked tissue change in a patient. Tests of all kinds may be within normal limits, and the only outstanding index of physiologic dysfunction may be marked loss of weight—thirty, forty or fifty pounds in one year, without recognized systemic disease. If tremendous loss of weight is combined with other systemic disorders, it must be considered as a definitely poor prognostic sign, especially in an individual in whom all forms of nutritional upbuilding have failed.

7. If this complete check-up of each patient is more than the otolaryngologist has time to carry out, then as the laboratory is used for its help, so the internist should be used, and the combined knowledge of both be brought to bear upon the problem. Often the one cannot work independently of the other.

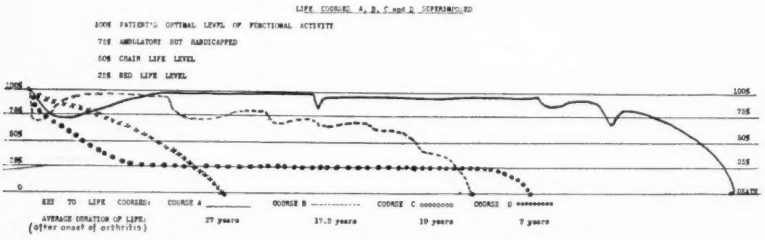
8. Lastly, if and when, tonsillectomy has been performed, each patient should be followed and watched just as carefully after as he should be before operation. Only in this way will it be learned whether the removal of the source of infection was of demonstrable value to that patient; whether it halted the progress of his systemic disease, or whether it added an additional load which he was unable to carry and so handicapped him further. Careful study of the individual, both before and after operation, is necessary if definite relationship between tonsils and systemic infection is to be proved, and the value of surgery versus conservative treatment is to be intelligently demonstrated.

My former ideas on this subject have been confirmed in the past few years by an intensive study of a group of about 500 arthritic patients.* Arthritis is a disease (and, in my opinion, one disease, though years have been devoted to fruitless discussion of types of arthritis) which is the epitome of all chronic disease. A group of arthritics represents the best possible single cross section of general systemic dysfunction. This study covered first a group of ninety-five who with death had completed a cycle of chronic disease.[3] Later a group of 400 living were similarly analyzed. These people, regard-

*The majority of these patients was followed at the Robert Breck Brigham Hospital in Boston from 1914 to January, 1934. The co-operation of the staff of the hospital enabled me to study the records and check on the patients. The assistance of Dr. Charles T. Porter, Chief of the Otolaryngological Service, was of particular value. He gave generously of his time and knowledge, so helping to complete the picture of the individual patient.

less of age, duration of arthritis or other systemic complication, followed four distinct courses from the cradle to the grave, and I believe all chronic patients would show similar life courses were they charted in like manner.

Course A represented a patient who with the onset of arthritis dropped to a lower level of activity; remained there for a time, then climbed back toward his former level and stayed at this level through the greater part of his remaining life. Course B showed a life course made up of definite periods of remission and relapse, and as the years passed, the trend was gradually downwards with lengthening and more frequent periods of relapse and shorter and fewer periods of remission. Course C showed a tremendous drop in the life line over a variable period of time, from the expected 100 per cent of activity to a 50 per cent or 25 per cent level, because of extensive



THE FOUR TYPICAL LIFE COURSES SUPERIMPOSED ON ONE ANOTHER.

joint destruction and crippling; and the remaining life spent at this low level, often for years. Course D showed a course which progressed steadily downward to death, without appreciable remission, regardless of treatment or any one particular factor.[4]

A detailed study of this group of arthritics from the point of view of the nasopharynx as a source of infection showed certain outstanding facts. At the onset of arthritic symptoms 10 per cent had had tonsillectomy performed; 90 per cent had retained their tonsils intact. Is this ratio of tonsillectomized patients to those with retained tonsils merely a normal incidence?

Does it indicate on the part of the average physician lack of recognition of chronic nasopharyngeal infection, particularly of the third and fourth types described?

Or does it indicate that the physician was given no opportunity to decide whether the nasopharynx was or was not a source of infec-

tion? The average patient in a public hospital ward is usually one without means, often with no great intelligence, and from a social stratum where such relatively minor body ills as repeated colds, repeated attacks of sore throat or tonsillitis occurring yearly or even more frequently are taken as part of the day's work and are allowed to continue without medical care. It is only when something new and incomprehensible is added to this accepted condition that medical aid is sought. By that time the spread of nasopharyngeal infection through the system has been accomplished, and in all probability it is too late to gain a great deal by removal of tonsils though they may have been a source of systemic infection years before.

In any case the mere fact that in a group of nearly 500 arthritic patients 90 per cent had retained their tonsils intact up to the time of the arthritic onset (and this group included patients fairly equally distributed in each decade of life) arouses one's interest in the speculation as to whether the arthritic picture would have been the same had the tonsils been removed in early childhood before the first attack of acute infectious tonsillitis occurred. Might such procedure have avoided future tonsillitis, so lessening extra load on other lymphoid tissue in the nasopharynx? Might it later in life have eliminated the possibility of tonsils being examined and considered as innocuous though in reality they were a definite source of infection missed by the examining physician because of lack of symptoms or complaints on the patient's part and lack of repeated observation of the nasopharynx on his own part?

TABLE I.

ENTIRE GROUP OF ARTHRITICS.

Life Courses	T. and A. Before Arthritic Onset	Tonsils at Arthritic Onset
Course A	17 per cent	83 per cent
Course B	7 per cent	93 per cent
Course C	11 per cent	89 per cent
Course D	18 per cent	82 per cent
Group followed five years or less	17 per cent	83 per cent
Total group	10 per cent	90 per cent

TABLE II.

TONSILS OUT BEFORE ARTHRITIC ONSET.

PERCENTAGE SHOWING RECOGNIZED JOINT REACTION TO NASOPHARYNGEAL INFECTION.

Life course A	20 per cent
Life course B	25 per cent
Life course C	66 per cent
Life course D	100 per cent
Group followed five years or less	50 per cent

The actual numbers in the above group were small (5, 14, 6, 2 and 10), but the disproportion of the results made the observation worth noting.

Fifty per cent had had recognized nasopharyngeal infections before and during their arthritis; 45 per cent had tonsillectomy after the onset of arthritis; 55 per cent have retained tonsils intact up to the present.

TABLE III.

TONSILS INTACT AT ONSET OF ARTHRITIS.

Life Courses	Tonsils Retained To Date	T. and A. After Arthritic Onset
Course A	50 per cent	50 per cent
Course B	53 per cent	47 per cent
Course C	59 per cent	41 per cent
Course D	61 per cent	39 per cent
Group followed five years or less	59 per cent	41 per cent
Total group	55 per cent	45 per cent

In course C only was there marked recognized association between joint reaction and nasopharyngeal infection. Of the twenty-nine patients in this course C, or the 59 per cent of course C group who have retained their tonsils intact to the present time, 41 per cent showed this definite reaction of the joints to nasopharyngeal infection, and in half the patients the reaction was tremendous and repeated.

CONCLUSION.

I am convinced that even without recognized association between upper respiratory tract infection and systemic (or more particularly joint) infection such association may be present. Proof of this depends on intensive study of the individual to determine three factors:

1. Recognition of obvious and concealed chronic infection in the nasopharyngeal tissue. The two most common are unmistakable, the acutely infected tonsil and the red hypertrophied chronic tonsil. The third is possibly of greater importance and is often missed unless the patient is seen frequently, and the appearance, subsidence and disappearance of the signs of infected lymphoid tissue are noted. On examination of this type of throat a purplish red discoloration is seen on the anterior tonsillar pillar, along the edges of the soft palate and at times in the uvula. These local changes are constant to a certain degree (the dusky discoloration being present at all times), but show varying reaction to exogenous and endogenous

infection. During such reactions the tonsillar poles become prominent and more inflamed, but this is noted only if the upper part of the tonsil is exposed. The duration of the reaction is short if the excitor is endogenous; long if of exogenous origin.

2. Recognition of single or multiple systemic infection. This depends on thorough physical, laboratory and radiographic examinations.

3. Determination of association between local and systemic infection by extensive and prolonged study of each individual patient. Long observation indicates that man follows roughly four natural life courses from birth to death. Study of a large group of patients with various chronic diseases has shown them to follow four life courses similar to the natural course but more exaggerated in their variations. These life courses have been termed course A, B, C and D. An individual following course A, after a period of lowered functional activity, returns to nearly his former level, and remains there practically until death. One following course B, from the onset of his disease until death, has periods of remission and relapse, with the life curve gradually sloping downward. A patient in the course C group, with the onset of his disease, drops in a variable period of time to a definitely lower level of activity and remains at this low level until death. In course D the individual's life course is comparatively short and the line shows a steady downward slant.

The ability to estimate the future life course of one's patient will prove of great value in determining the best procedure to adopt when nasopharyngeal infection is present. Generally speaking, if a patient presents himself with combined nasopharyngeal and systemic infection, having had no operation performed on the tonsils, I believe the lesser of two evils—i. e., removal of tonsils as a possible focus of infection, with the risk of disaster, or leaving them intact as a constant source of infection via drainage with equal possibility of disaster—is removal. Success or failure following such procedure depends on the time chosen for operation. If good chance (or good judgment) makes the time tonsillectomy is performed opportune, the result will be beneficial to the patient. This is the point on which attention should be focused, and more certainty gained as to this crucial time. Greater effort should be made to determine the optimal time for removal.

In an arthritic it should be performed when both the nasopharynx and the joints are as quiescent as possible; when the patient's resistance and general health have been raised to the optimal level,

and only if the patient shows periods of such quiescence and measurable improvement. In an arthritic who appears never to reach even a short period of quiescence of joint disease, or of general improvement, who is markedly undernourished and who cannot be made to gain weight, I believe tonsillectomy is never indicated and that the only practical way of keeping nasopharyngeal infection in check is by whatever local treatment can be used. This "hands off" policy is also indicated in any individual, whatever his disease, who is believed to be following life course D. Any operative interference on one in this group appears to be disastrous.

Wise decision between operation and conservative treatment of nasopharyngeal infection depends wholly on the individual patient in question. Without a thorough knowledge of him and of his physiology the outcome is pure chance, and science remains without proof of the relationship between tonsillar and systemic infection.

205 BEACON STREET.

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XVIII.

TERTIARY SYPHILITIC INFILTRATIVE LESIONS OF THE NASAL MUCOSA.*

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Tertiary syphilitic lesions of the nose present an ever present problem to the otolaryngologist—first, because of the prevalence of the disease, and second, the difficulty of early diagnosis in manifest syphilis as it passes from the state of latency to activity. Syphilis of the third stage shows a marked predilection for attacking the various constituents that make up the structure of the nose. Behaving here as elsewhere, syphilitic lesions may undergo absorption if recognized and properly treated, or may spread rapidly to the surrounding and underlying structures, producing a destructive necrosis with resultant deformity. In a general review of this subject of specific infiltrative lesions of the nasal mucosa, it will be necessary to have a definite knowledge of what is meant by the term latent syphilis. With this object in mind we will briefly review some of the accepted fundamentals of syphilis as a whole, from the entrance of the spirochete into the human host to that quiescent late stage described as latent syphilis. Herein we shall rely on the statements of such accepted authorities as Stokes[1] and Warthin.

The older conception of initial infection was that syphilis was a local rather than a systemic infection; that it remained a local condition for some time and then, by a sudden eruptive phenomenon or outburst, spread and was transformed into a general or constitutional disease. However, this is not the case; the chancre is rather a late manifestation and is merely a tardy local reaction at the point of entry of an infection which presumably has become systemic long before its existence has become suspected or demonstrated.

Immediately after infection the first of the natural defenses assumes its rôle, namely, a local pathologic mechanism, which from the standpoint of development of immunity and occurrence of relapse is quite important. The syphilitic chancre or primary lesion

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which develops at the point of entry of the organism is the prototype of millions of local reactions occurring throughout the body during the course of a syphilitic infection.

The first step after penetration is an invasion of the perivascular lymphatics by the spirochete, followed by a vascular reaction which assumes the form of an endothelial swelling with proliferation and development of an obliterative endarteritis. This is accompanied by perivascular infiltration with lymphocytes, the appearance of plasma cells and fibroblastic proliferation and healing. Immediately following invasion and during the phase of vascular and perivascular reaction the spirochetes increase rapidly in numbers. Penetration into the surrounding lymphatic channels and the escape of the rapidly reproducing invaders into the blood stream occurs. Then a change takes place as the histologic signs of healing begin to appear. Fragmentation of the spirochetes may be seen, the number and viability of the organisms decline and evidently, under the influence of an as yet uncomprehended biochemical mechanism, the focal infection or chancre subsides.

If the local reaction which inhibits or destroys the spirochete in each local lesion or focus would entirely perform its duty, immediate and spontaneous cure of the disease would result. Unfortunately the successive inoculations of adjacent and distant tissues produced by the multiplying and escaping spirochetes prolongs the disease almost indefinitely in time. Thus we see that the local defense mechanism may completely destroy, in part or in whole, the invading organism. It may destroy their virulence and reproductive powers, though not, however, necessarily disintegrate them. Even though the activity of the surviving organisms at the site of a healing lesion may be temporarily suspended, sooner or later some degree of relapse occurs. The local defense mechanism which has developed loses its effect, the tissue immunity wears off, and reproduction of virulent organisms occurs, followed by reinoculation of the local lymphatic area or even of the blood stream. A fresh reaction in newly infected tissue begins and syphilis resumes once more its cycle of infection, reproduction, suppression and relapse.

Fortunately for the victim of the disease, the description of the local reaction or defense to the spirochete in the tissues does not complete the story of immunity processes which defend the body against this infection. The systemic or general defense in syphilis stands between the patient and the ultimate complete involvement of every cell in his body in endless cycles of infection, reaction and relapse.

Various forms of systemic defense have been described, among which are the development of antitoxins; however, no such antitoxin of the extracellular or circulating type has been developed to the spirochete, so far as has been ascertained. A certain amount of agglutinin for the spirochete appears in the blood stream of the syphilitic patient, but this feature of his defense mechanism is insufficient to explain all the phenomena. Lysins and substances which stimulate phagocytosis are also formed to some extent. It is probable that the tissue defense of histiocytes is very early called into action, during the acute stage; but these reticulo-endothelial elements are rapidly exhausted, yielding to the plasma cells and themselves disintegrating, choking the lymph channels or metamorphosing into fibroblasts.

Among the most important factors concerned in the systemic or general defense against syphilis is that of the chronic inflammatory defense described by Warthin.[2] The long periods of latency or that period of suspended animation of the spirochete which marks the clinical course of syphilis are products of a relative immunity maintained by slow chronic inflammatory changes, especially in the parenchymatous structures and the vascular system. The spirochetes lying in the lymph spaces around the capillary vessels are surrounded by a group of lymphocytes and plasma cells. which collect at this site as the result of an irritative reaction. This reaction set up in the vessel is in the form of an endarteritis and reduces the blood supply of the dependent part which it supplies, with ensuing degenerative changes. The lymphocytic infiltration, following identically the course of the typical reaction at all stages of the disease, is gradually replaced by fibroblasts, and fibrosis with further loss of parenchyma results. These minute microscopic reactions just described are infinitely numerous. Their slow combined inflammatory reaction functionally impairs and destroys the very substance of active parenchymatous tissue, replacing it with inert and weakened scar tissue. This chronic inflammatory reaction in microscopic foci keeps the spirochete in subjection and the patient is in the clinical state of latency. As in the early active focus, the organisms in any local focus may be entirely destroyed and fibrosis may result, or they may be held in an inert state, nonproductive and nonpathogenic. Some authors would lead us to believe that the spirochete has a resting stage. If, however, the spirochete's vitality is superior to the lymphatic and vascular opposition, they may begin to reproduce and become virulent, and a large focus may develop into a true internal chancre, from which spirochetes may be distributed to the blood stream to start a relapse years after the primary infection has been suppressed spontaneously or by the effect of treatment.

An additional type of defense, namely, the allergic defense, is the most doubtfully effective of the reactions of the host against the spirochete. It appears to be an explosive inflammatory reaction, not a defense, with necrosis of the tissues in contact with a relatively small number of spirochetes, and usually occurs late in the course of the disease. This reaction is in reality a fulminating local collapse of the tissue involved. It must be kept in mind that in early syphilis the number of organisms in the tissues and circulation is extremely large, yet the local reactions are comparatively mild. In the late stages of syphilis the number of organisms is greatly reduced, due to the various resources of the defense mechanism and treatment. Gumma, the characteristic late lesion, presents an extremely small amount of detectable organisms; yet the tissues involved show more actual destruction and fibrosis than the early type of lesion. It may be possible that the disintegrating, dead or dying organism, or even the toxic products of dead spirochetes may produce some distinctive tissue reaction characterized by local violence. As pointed out by Neisser, Finger[9] and other observers, there is probably some other condition than the presence of a few spirochetes necessary to produce the violent explosive reactions seen in the late syphilids. This has been generally accepted as a hypersensitiveness of the tissue, which leads it to react in such a violent manner. Other factors of an extrinsic nature, such as chronic vascular degenerations, may so reduce the general resistance of the affected parts that a small number of organisms may produce havoc and destruction not possible in normal tissue.

The gumma constitutes a distinct type of reaction, as does the microscopic inflammatory focus; in fact, the two are differentiated by degree rather than kind. The microscopic focus is in reality a military type of gumma. There is a stage between these two conditions which presents a marked round cell infiltration, which may be termed that of pregummatous infiltration. Large areas of lymphocytic and plasma cell infiltration develop rapidly, with an extensive peripheral endarteritis and obliteration of vascular supply that may lead eventually to massive central necrosis. Later, the growth assumes the characteristics of a granuloma, and giant cells appear. Peripheral organization may occur and healing may result in diffuse fibrosis rather than central softening. If the lesion is near the mucous surface a central slough of necrotic tissue forms and breaks down, leaving a crateriform ulcer.

During the latent period of syphilis the mucosa of the nose, as elsewhere, may show no macroscopic changes. When some condition arises which may disturb the mutual tolerance developed between the

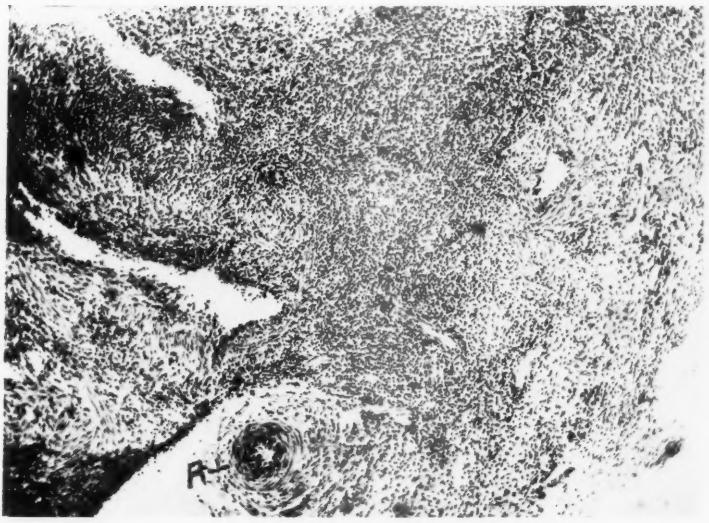

Fig. 1.

spirochetes and the tissue in which they lie, changes become apparent. Fortunately the activation of latent syphilis does not occur simultaneously in every part of the host. Syphilis assumes strange rôles and upon occasion may be called "spotty." Although the patient may present microscopic lesions of latent syphilis in every portion of the body, it appears that activation of the process may select one individual site to display its specific activity, as in the case of infiltrative nasal mucosal lesions with apparently no evidence of clinical activity elsewhere, completley ignoring the bony or cartilaginous constituents of the nose, or vice versa. However, as the process continues, extension usually occurs from one structure to the other. Other notable examples of this selective peculiarity of the disease include discrete gumma of the iris, acute labyrinthitis simulating Ménière's disease, and acute perichondritis of the thyroid cartilage, epiglottis or arytenoids.

From observation of individuals with apparently normal mucous membranes in cases of known latent tertiary syphilis in which the disease has for some reason become manifest, we have observed the relative frequency with which the intranasal mucosa has been involved as compared to that lining the accessory sinuses. The accessory sinuses, as their name implies, are associate anatomic structures, not of essen-

tial physiologic value to the human species. The vascular and lymphatic formation of sinus mucosa is accordingly rather elementary as compared to that of nasal mucosa. That is, we find the most prominent development of these structures, so important in syphilis, in the direct airways of the nose. A possible explanation for this predilection would seem to be suggested by this difference of vascularization of the two types of mucosa, coupled with the fact that the lesion of latent syphilis is predominantly vascular and perivascular. Normal comparative histology of these membranes impresses us with the preponderance of vascular elements in the mucosa of the turbinate bodies and nasal passages proper; and these factors determine the likelihood of a luetic attack upon this mucosa. Henke and Lubarsch[1] state that the syphilitic process may start from the mucosa or bone, and that apparently the mucosal onset is more frequent; this being penetrated by a diffuse infiltration which consists of round cells with large nuclei, and succeeds in penetrating not only the mucosa and periosteum, but also the bone itself. For this reason, according to Lieven,[5] the nose forms sharply circumscribing gummas much less frequently than other organs. One may see under the microscope almost always that the neighboring and apparently slightly congested parts of the mucosa already show specific changes, while macroscopically we see only a limited nodule. Such conditions are found mostly on the floor of the nose and in the septum and may be of considerable size. The nasal mucosa may be considered as a target for continued selective trauma, as evidenced by the preponderance and variety of infections and extrinsic factors that select this site for their activities. General physical states that lower body resistance play an important rôle in favoring infection by reduction of local resistance in the upper respiratory tract. In fact, any condition, either local or general, which may succeed in disrupting the balance between the tissues of the nose and the spirochete, is capable of activating syphilis which may be said to be in a clinical state of latency, giving the organisms lurking in and about the vascular elements of the lining mucosa a fresh impetus.

It is utterly impossible to define any set group of symptoms which are absolutely characteristic of beginning nasal syphilis. The "specific cold" is absolutely without differentiating symptoms or signs as compared with the common cold at its inception. However, as the disease progresses certain characteristics are practically so constant in occurrence that they simulate a clinical entity. Among the first of the findings is the unresolved head cold, progressing to a point where it becomes almost a permanent disability in so far as the patient is

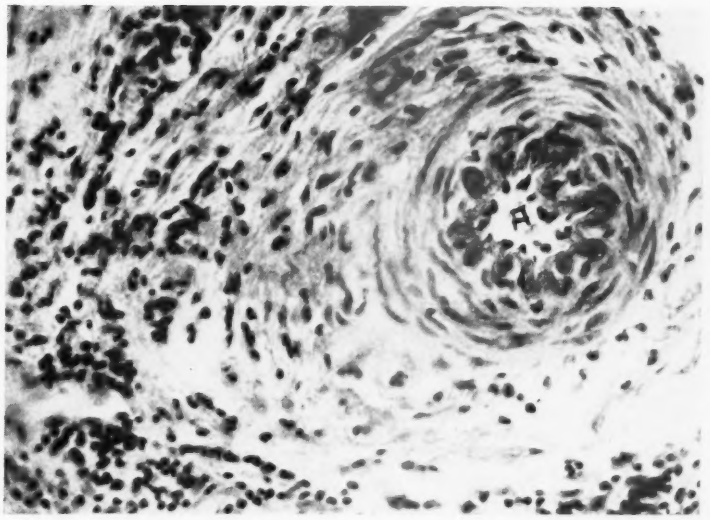

Fig. 2.

concerned. It is at this stage that the predominant symptom of impaired breathing usually brings the individual in for investigation. The nasal obstruction is constant and severe, and the patient does not have the periods of relief from this obstruction that occur during an acute rhinitis or even a sinusitis. This phenomena is easily explained by the fact that the process causing this obstruction is in reality an induration of the mucosa, the tissues being packed with round cells forming an integral part of the mucosa, in contradistinction to a transitory inflammatory edema found in nonspecific infections. Nasal discharge is scant at first, but later appears in various amounts and after a time becomes more or less purulent and bloody, with the advent of coexistent sinus involvement. Small crusts form and the sense of smell may be impaired or absent; some observers call attention to the quite characteristic musty odor emanating from the noses of these patients.

Inspection of the mucosa reveals an indurative swelling which may be present on one or both nasal passages. It may be generalized, involving the entire area of the lining mucosa, or localized to some restricted area. Observation has revealed that lesions affecting the mucosa alone have a predilection for invasion of the vault of the nose, the lateral nasal wall (including the turbinate bodies) and the anterior portion of the septum. Lesions affecting the floor of the nose

and the entire septum usually suggest deeper involvement, not only of the mucosa but the periosteum, bone and cartilage as well.

The luetic membrane may be described as having a dusky submucosal flush of bluish red color as contrasted with the superficial angry scarlet of an acute infection. Its appearance suggests induration and an impediment to the outflow of venous blood. As the condition progresses the color of the mucosa loses its definite hue and becomes a dirty yellowish white, suggesting even more induration and interference with the arterial blood supply as a result of increased endarteritis, further crowding of the area with round cells and beginning fibrosis with resultant local anemia. To the touch the membrane has an elastic, firm feeling. Any of the various shrinking agents, such as cocain or adrenalin, do not shrink this membrane to any appreciable extent and do not cause the peculiar color to disappear. Jacques[6] has discussed the diffuse polypoid elevations, apparently granulomatous in type, found all over the ethmoid area: vegetations, grayish yellow in color but distinguished from ordinary polypoid degenerations by their diffuse location, relatively firm consistency and lesser mobility. Confusion often arises at this stage; allergic conditions are often suspected. Often the individual has some allergic reaction to the various tests devised and he may be subjected to weeks and even months of desensitization.

Involvement of the sinus mucosa may occur to a greater or lesser degree, often as a pansinusitis. The sinus involvement may be primary in the sense that the activated latent lesion may have its inception in the mucosa of the sinus itself. It may also be involved by extension and tissue continuity from intranasal lesions or from lesions involving the bony or periosteal structures surrounding the sinuses or adjacent to them. The production of a nonsyphilitic sinusitis due to insufficient aeration and drainage and the invasion of nonspecific bacteria, is the most commonly associated type of involvement. Frequently, however, as suggested by Dr. Joseph Beck,[7] sinusitis may be the result of a vascular block and breaking down, especially noticed in the ethmoid labyrinth, although he has never been able to demonstrate it in any of his preparations of ethmoid exenteration in syphilitics.

The symptoms and signs produced by sinus invasion are those characteristic of the sinus involved. Graham[8] has observed that by x-ray examination the sinuses have a blotchy appearance with scattered light areas and that pus is not necessarily present. He also states that sinus tenderness may be entirely absent. Periosteal thickening is present and usually is permanent. He also feels that this might ac-

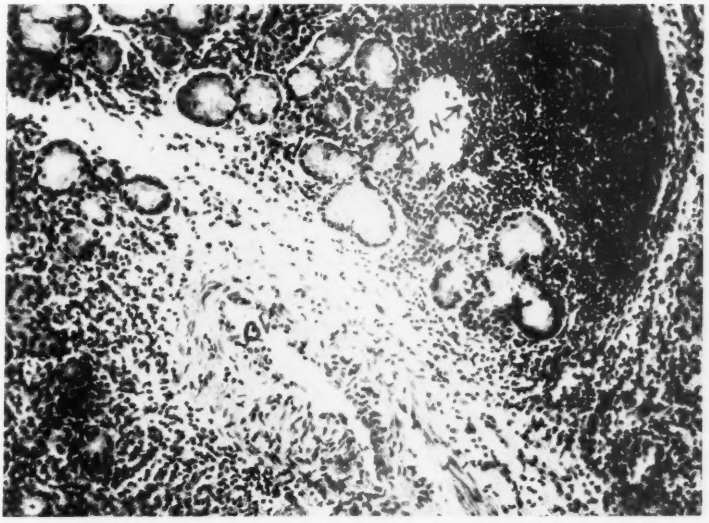

Fig. 3.

count for many of the plates which later show apparent sinus involvement but fail to demonstrate it under operative exploration.

Pain[9] is more or less irregular in character, more often absent, but headache is common, as referred from the sinuses and nerve lesions involved, particularly nocturnal pain frequently associated with earache. The nerve[8] lesions are those involving the olfactory, involving loss of smell, and those of the trigeminal and vasomotor nerves. Pain over the bridge of the nose is frequently present and may at times be quite severe.

Warthin[10] states that the ultimate diagnosis of syphilitic infection lies with the microscope. As to correlation between the Wassermann reaction and pathologic findings, negative reactions are very common when the microscopic findings are positive; therefore a negative Wassermann has very little value in excluding the presence of latent syphilis. He also states that the Kahn test has shown a much greater degree of accuracy of correlation, particularly the so-called "presumptive Kahn test," which has shown few errors in checking with autopsy findings.

The question arises as to the possibility of demonstrating the spirochete in sections of the mucosa removed for purposes of biopsy.

Wile[11] states that spirochetes can be demonstrated, particularly in those forms of syphilitic rhinitis which characterizes the florid lesions of postnasal syphilis, either acquired or congenital, and that spirochetes are demonstrable only with great difficulty in the infiltrative stage of gummous involvement. So far as he knows, this has not been done in gummas involving the nasal bones and soft tissues, for the reason that it would require complete excision of such a mass to make proper serial sections. Beck[7] states that he has never seen the spirochete within the tissues but only in the dark field in secretions.

Biopsy, however, of a small bit of tissue removed from the most active portion of the mucosal lesion in questionable cases is of extreme value to the clinician. In[12] the presence of lues it will usually show the typical histologic elements of a syphilitic lesion, such as round cell perivascular infiltration, various degrees of endarteritis and numerous plasma cells which may be said to be the predominant cell of syphilis. Should the section show at least presumptive and supportive evidence of a chronic luetic rhinitis, it would have served its purpose well indeed by arousing suspicion of a specific background. The otolaryngologist should have as part of his equipment a knowledge of the cutaneous lesions and the scars of late syphilis, which if correctly interpreted, may identify a syphilitic infection which would otherwise be missed because of negative serology or atypical signs. Careful physical examinations, minute history taking extending far into the past and the aid and co-operation of a well trained syphilologist are indispensable and invaluable in the proper interpretation or final analysis of late syphilis.

There are,[13] also, combinations of syphilis with tuberculosis; such cases are mentioned by Lindt and Finder. There is little difficulty in differential diagnosis between lupus and tertiary syphilitic ulcers with sharp and steep walled margins and strong surrounding reaction, characteristic gummas. Ulcers with flabby granulations and rather slight surrounding reaction are found in lupus; in later stages near the ulcerated spots fresh nodes shoot up with atrophic scars visible between them. In spite of this difference in typical cases, the diagnosis may be very difficult; especially in a form which Bindermann has described as diffuse hypertrophic syphiloma from diffuse infiltration of the nasal septum and the alæ nasi. At first, flat ulcers arise which become covered with crusts. There arises here, as in lupus, a disposition of the base and edge of the ulcer to papillary growths, so that infiltration occurs near by. Further differential diagnostic difficulties arise from small gummata among the scars, which look very much like lupus; this can only be worked out serologically.

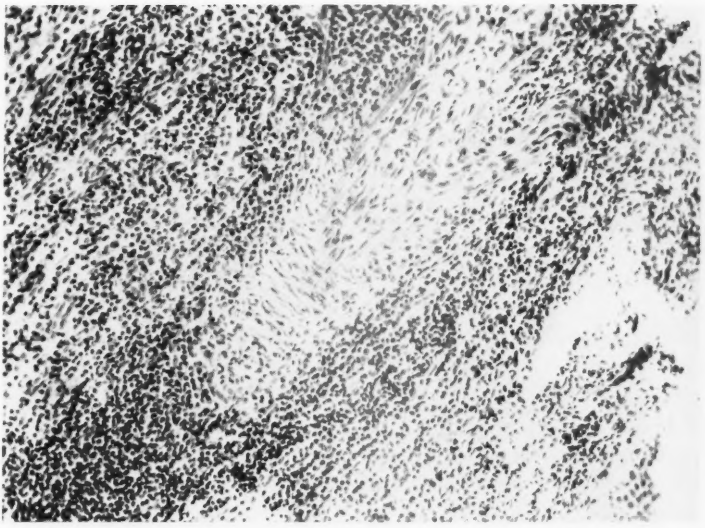

Fig. 4.

Also, where there is no certain history of syphilis, cases may be seen in which both sides of the nose are filled up with granulomas resembling sarcoma. As to rhinoscleroma, we must remember that this very chronic infiltration takes an extremely long time to develop and ulcerates late, finally leading to a shrinkage of the tissues.[11] In extremely difficult cases microscopic investigation will show Mikulicz cells and the bacilli of rhinoscleroma.

Microscopic sections were made from a small bit of mucosa in a case presenting confusing nasal symptoms over a period of four months, during which time the individual had been subjected to various kinds of treatments, three months of vaccine therapy and a tonsillectomy. Her physical examination was essentially negative, and the blood Wassermann negative on several occasions. However, examination of the spinal fluid revealed a four plus positive luetic reaction and the marked improvement of the condition under specific therapy leads one to the conclusion that the evidence of pathologic changes described, although not conclusive, is at least presumptive and supporting evidence of a chronic luetic rhinitis. The following photomicrographs show the changes found in the above mentioned case:

In the more superficial zone of the mucosa there is a marked cellular infiltration about blood vessels (capillary veins and arterioles) which are distributed in a manner to indicate that they constituted a part of a pattern of granulation tissue (Fig. 1). The cells, which consist of varying numbers of lymphocytes, plasma cells and large mononuclear phagocytes, vary in their distribution and concentration within the fibrous connective tissue stroma that surrounds the blood vessels. The fibrous connective tissue also varies markedly in its character from young newly formed fibroblasts to old, dense scar tissue accumulations that are less markedly infiltrated (Fig. 5A). There appears to be some relationship between the cellular infiltration and the walls of blood vessels. This is particularly noted in relationship to the smaller arterioles. In the latter there is evidence of a mild obliterative endarteritis and the perivascular zone of the adventitia discloses a definite accumulation of fibroblasts and lymphocytes that encircle the arteriole to form a more or less definite collar. In these perivascular lymphatics and tissue spaces plasma cells and lymphocytes as well as mononuclear phagocytes are found. The wandering cell infiltration here is not unusually pronounced but is sufficiently striking to indicate that there is a perivascular inflammatory process (Figs. 1 and 2A). It is also observed that about the walls of veins that are cut at right angles or obliquely there is an accumulation of lymphocytes and plasma cells that seems to parallel the course of the vessels (Figs. 4 and 5). In number of areas the walls of rather large veins are infiltrated throughout by lymphocytes, plasma cells and other wandering cells. The proportion of wandering cells and scar tissue varied markedly. Here and there a somewhat atypical giant cell may be seen (Fig. 5G). About the mucous glands similar changes as described above are noted in relationship to the blood vessels, but the accumulation of lymphocytes is much greater, often forming so-called pseudolymph nodes (Fig. 3L. N). In the sections examined there is no evidence of discrete tubercle formation nor are there any areas of caseation necrosis.

The entire inflammatory process in the nasal mucosa is a proliferative one, partaking of the nature of a chronic granulomatous process which has progressed without marked degeneration but with a considerable disturbance in the structural pattern of the nasal mucosa. In evaluating the pathologic changes that are present in this mucous membrane it is necessary to point out several outstanding characteristics, namely, (1) the richly cellular infiltration consisting of plasma cells, lymphocytes and mononuclear wandering cells. Polymorphonuclear leucocytes are not present; eosinophiles are sparsely

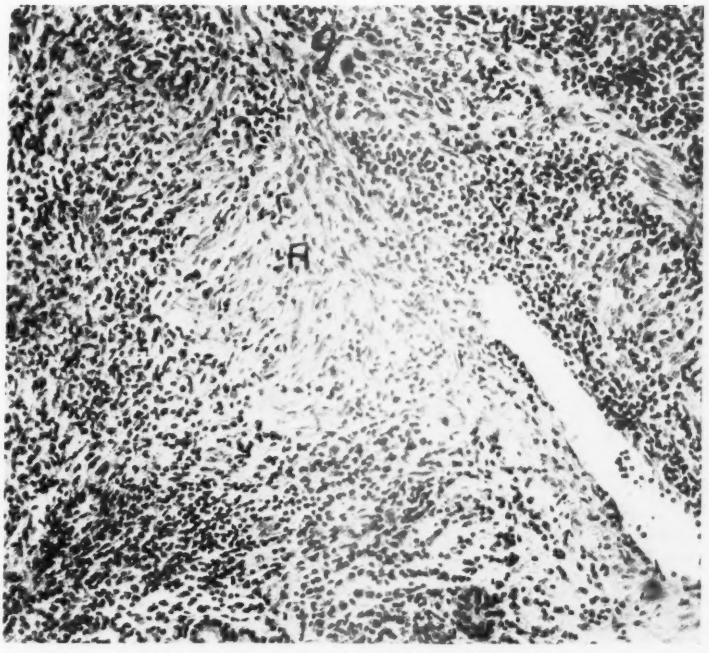

Fig. 5.

scattered; (2) the occurrence of scattered giant cells; (3) the abundance of fibrous connective tissue, and (4) the association of these changes about the blood vessels and in particular the perivascular lymphatics. All of these changes may be construed as presumptive evidence of the existence of lues. The absence of a marked obliterative endarteritis in the section examined may be considered as evidence against the conclusion that a treponema pallida infection is responsible for the pathologic changes.

With respect to the plasma cell response, we agree with the conclusions of Lederer, Fabricant and Milles,[15] that such response accompanies many sorts of chronic septic processes. In syphilis, however, there is little or no eosinophilic infiltration, and an especially rapid fibrosis. Similar plasma cell proliferation was extensively studied by Miller,[16] using tuberculoprotein intraperitoneally. Clinical appearance of tubercular lesions in the nose will, however, readily distinguish them from the diffuse infiltrations of syphilis.

SUMMARY AND CONCLUSIONS.

1. Syphilitic infiltration of the nasal mucosa is a definite clinical entity and should be considered in the differential diagnosis of every doubtful lesion of the nasal mucosa.

2. Because of the preponderance of vascular and lymphatic elements present in the mucosa of the direct airways of the nose, specific infiltrative lesions are more frequently found here than in the mucosa lining the accessory sinuses.

3. Biopsy of the nasal mucosa is of extreme value to the clinician in questionable cases. Should the section show at least presumptive and supportive evidence of a chronic luetic rhinitis, it will have served its purpose well by arousing suspicion of a specific background.

4. Negative blood examinations should not preclude the possibility of a specific infection in questionable lesions of the nasal mucosa and in doubtful cases one should resort to careful study of the spinal fluid.

MEDICAL ARTS BUILDING.

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XIX.

MICROTIA WITH MEATAL ATRESIA, WITH DESCRIPTION OF AN OPERATION FOR ITS CORRECTION: REPORT OF TWO CASES.*

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Of the various congenital anomalies of the ear, microtia with meatal atresia is relatively rare but occurs much more frequently than is generally supposed. Because of the commonly accepted opinion that therapy offers relatively little, these cases have not previously received sufficient attention.

There is no uniformity of opinion concerning the therapeutic value of operative procedures. Of the various operations, radical mastoidectomy has been most frequently employed. Ruttin, Dahmer, Havach, Urbantschitsch and Mosher report considerable improvement in cases so treated, whereas Bruehl and Blumenthal found no improvement in hearing after operation, although functional examination showed an apparently normal labyrinth. Scheibe grafted the mastoid wound with skin with good results. Alexander, on the other hand, maintains that operation should be performed only if the patient has otitis media and mastoiditis on the affected side, and that improvement in hearing is not sufficient to justify the operation in unilateral involvement.

Based upon the fact that the labyrinth is formed embryologically from the otic vesicle before the development of the external and middle ears, one of the authors (R. H.) has been of the opinion for some time that impairment in hearing in these cases is due largely to obstruction to the transmission of sound waves. The finding of normal labyrinths in the study of microscopic sections of autopsy material in cases of microtia with meatal atresia substantiates this contention. If this premise is correct, a successful operative procedure would permit the direction of the sound waves to a properly receptive portion of the labyrinth. This contention is further substantiated by

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the fact that an old type ear trumpet collects and directs the sound waves to a possible remaining portion of the sound apparatus. Fletcher and others demonstrated experimentally the soundness of these convictions.

If the above opinions are correct, it is rational that the ideal treatment consists of the construction of an external auditory meatus in correct anatomic manner in order that sound waves can be directed efficiently to the receptive portion of the middle ear. The treatment of these unfortunates by radical mastoidectomy does not fulfill this requirement and is not justified because the procedure is anatomically and physiologically incorrect, in that the opening of the canal is formed posterior to the external ear, which normally serves as a mechanism for collecting sound waves (as in the old style ear trumpet). The relatively slight and uncertain results obtained by this operation do not justify such a radical procedure. From the anatomic investigations of Fraser and our own roentgenologic examination of cases with microtia and external auditory meatus atresia, it is evident that in many of these cases a bony canal exists. We believe that in these cases the lining of the bony canal with skin and construction of a canal extending from the external surface to the internal ear, in the position of the normal external auditory canal, is theoretically and practically ideal. Such a procedure has many advantages over the radical mastoidectomy and other operations. It is by far the simplest, is attended with the least surgical risk, and can be more quickly performed than any of the other procedures. In case radical mastoidectomy should be elected subsequently, the construction of a canal anterior to the ear would facilitate the more radical operation and would serve as a preliminary stage to the radical procedure.

The technic of the operation for the construction of an external auditory canal in microtia with congenital atresia, as proposed and performed by the authors, is as follows: A curved incision with the concavity anterior is made posterior to the tragus, the midportion of which is placed in the depression of the skin located in the position of the normal external auditory meatus. After incision is made through the skin, the bony external auditory canal is located by careful blunt dissection and followed backward and inward along its course by separating and dissecting through the dense fibrous tissue which fills its lumen and is firmly adherent to the canal wall. No difficulty is encountered from hemorrhage, for the area dissected is essentially avascular and the field remains practically bloodless. The dissection is carried along the canal to the depth approximately equal to that of

the normal external auditory canal. At the depth mentioned we found in the two cases herein reported a definite resistance which was not unlike bone. We were unable to determine, because of the fear of excessive trauma, whether the resistance was a very dense fibrous tissue membrane or a thin bony plate, either of which could serve as a sounding board. We are inclined to think it was the latter.

Immediately after the dissection of the canal a "stent" of the cavity is made of a suitable dental compound, flaring it slightly in the external portion in order to insure ample opening at the skin surface. A Thiersch graft is then removed from a hairless area on the thigh. After applying the graft to the "stent" (skin surface adjacent to the "stent"), it is inserted into the newly formed external canal and held intimately fixed in position by means of retention sutures of silkworm gut. A dry dressing is applied and not disturbed for approximately ten days, at the end of which time the sutures are removed, the "stent" withdrawn and the graft examined. Hearing tests can be made at this time. In order to prevent contraction of the graft, the "stent" should be worn held fixed in its original position by a small ear bandage, for a period of eight to ten weeks. Dressings are done regularly, at which time the canal and "stent" are cleaned. The massaging action of the "stent" on the graft serves to make the skin lining more pliable. Following the removal of the "stent" there is no tendency for the canal to close or is there any unsatisfactory change in the graft. Patients are instructed to keep the canals clean by the application of warm water and soap and to prevent excessive dryness by application of a bland cream when indicated. The above described operation has been performed successfully in two cases, the reports of which follow:

REPORTS OF CASES.

CASE 1.—A white woman, aged 26, was admitted in September, 1934, with the complaint of partial deafness associated with a disfigurement of her right ear. This condition had been present as long as she could remember. There was no history of any member of her family having had a similar complaint. Nothing of further importance was elicited from her past history. The patient's husband stated that she was rather shy and of a retiring nature. Physical examination revealed nothing of importance except microtia of the right ear associated with a congential meatal atresia. The left ear was well formed but showed a stenosis of the external auditory canal. Examination of the hypopharynx revealed that the eustachian tubes were apparently patulous with no evidence of abnormality. Roentgenologic examinations showed the mastoid cells to be pneumatized and a formed auditory canal in the bony structure. A hearing test revealed that the patient experienced difficulty in hearing the lower tones; all higher tones were heard perfectly. The lowest tone which the patient could hear was from a C-3 tuning fork (1048 D.V.) Bone conduction was good.

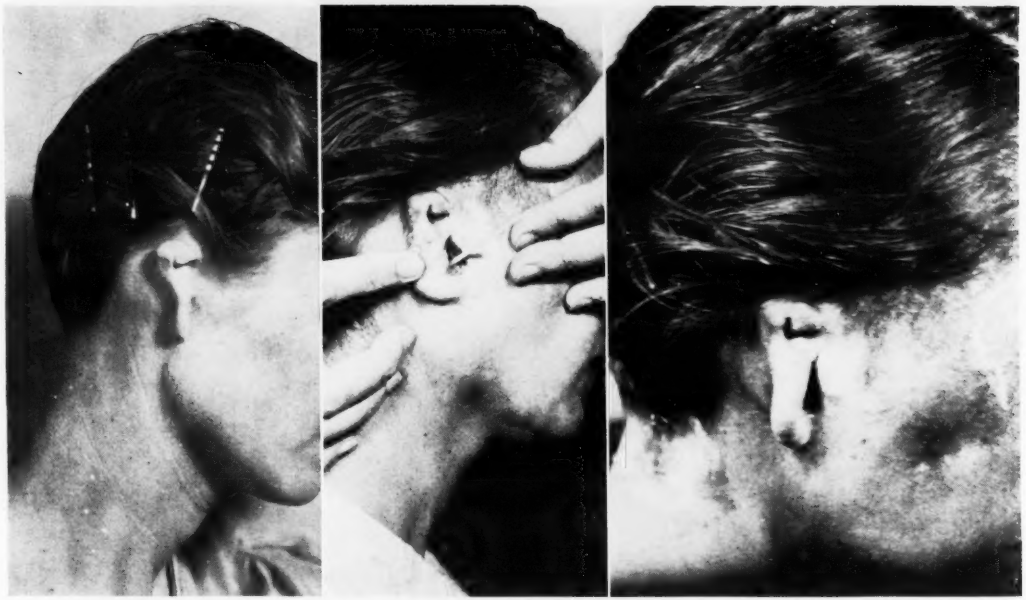

Fig. 1. Fig. 2. Fig. 3.

Fig. 1. Photograph of patient described in Case 1, showing the type of microtia and the absence of any external auditory meatus. Note the unusual appearance caused by the absence of an external auditory canal.

Fig. 2. Photograph showing the results of the operation. The lobe of the ear is pulled backward to give an idea of the size of the opening made, and a toothpick is inserted in the canal in order to show depth.

Fig. 3. Photograph of patient following operation, showing the improvement in the general appearance of the patient after an external auditory canal has been established.

The patient was operated upon according to the technic described and at the end of nine days, when the "stent" was removed, the graft was seen to have taken satisfactorily. Hearing tests done at this time showed that she could hear satisfactorily the lowest tuning fork—the C (128 D.V.).

This patient returned recently for observation about three months after operation, and at this time hearing tests revealed that all tones previously heard were equally audible and that conduction time was increased. Patient's husband stated that there was a marked improvement in the auditory reception and response to the spoken voice. The patient herself had gained noticeably in weight and showed an enthusiastic spirit which she previously had not manifested. There was no tendency for the newly formed external auditory canal to close. There was improvement in the general appearance of the ear, the new canal giving it a more normal anatomic appearance than that present before the skin posterior to the tragus had been perforated.

Fig. 4.

Fig. 5.

Fig. 4. Photograph of patient described in Case 2, showing the type of microtia and also the absence of any external auditory meatus. The absence of the external auditory canal in this case is not so pronounced as seen in Case 1.

Fig. 5. Photograph taken after operation, showing the size of the cavity which resulted.

CASE 2.—A white female child, aged 7, was seen in consultation with Dr. L. W. Alexander, with a chief complaint of impaired hearing associated with a malformed right ear. The child showed marked impairment in hearing and had developed the habit of turning the head toward the left side where reception was better. There was such marked diminution in hearing that her parents had placed her under the care of a lip reading instructor. The family history was negative for similar lesions either on the maternal or the paternal side. Physical examination was essentially negative except for a pronounced microtia of the right ear with a congenital meatal atresia. There was a slight stenosis of the external

auditory canal of the left ear. There was a noticeable shortening of the palpebral fissure at the outer canthus of both eyes. Hearing tests showed that reception was good for the higher tones but absent for the lower. A C-3 (1048 D.V.) tuning fork was the lowest heard by this child. Bone conduction was good. No examination of the eustachian tubes was possible due to lack of co-operation by the child. Roentgenologic studies showed a fair pneumatization of the mastoid cells and the presence of an osseous canal.

The above described operation was performed on this patient. The "stent" was removed at the end of nine days, at which time the graft was found viable. Because of lack of cooperation by the patient the first dressing was done with the patient anesthetized by nitrous oxide and oxygen. Hearing tests were therefore not possible at the time of the initial removal of the "stent" but were performed three days later. The test following operation showed that in contrast to pre-operative observations in which the lowest audible tone was 1048 D.V. (C-3 fork), tones as low as 128 D.V. (C fork) could be heard.

The child has been under observation almost three months since operation and there is no tendency for the canal to close. The hearing is still improving, and at the present time she can hear a moderately low spoken voice at a distance of seven feet, with the mouth shielded to prevent lip reading. There is also cosmetic improvement in the ear, due to the presence of an opening at the usual site for the external auditory canal. In addition, there is a noticeable improvement in the child's general interest, which followed the improvement in hearing.

Based upon the results obtained in the two cases reported above and the extreme simplicity of the operation, we feel that we are justified in advocating this procedure in cases of unilateral congenital microtia and meatal atresia in which there is marked impairment in hearing. We are in accord with the feeling that the procedures previously done, involving radical maneuvers, should be limited to bilateral cases, for here the risk is grave and the ultimate result is questionable. Even if the radical operation is elected, we feel that the technic proposed is a logical preliminary procedure, for an external canal will be established with a stable cutaneous lining. This would make a second, more radical stage, less formidable and consequently attended with less risk.

We feel that the following are requisites for the election of this procedure: (1) Normally pneumatized mastoids as shown by x-ray; (2) the loss of hearing limited to the lower tones, thus establishing the fact that the conductive mechanism is at fault; (3) normal hearing for the high notes; (4) normal or slightly increased bone conduction, and (5) patulous eustachian tubes.

From the observations in the two above cases we believe that operation should be attempted relatively early, for two reasons: (1) Patients unoperated upon until after adolescence are subjected to the definite risk of nerve atrophy; and (2), patients with impaired hear-

ing for a long period of time develop the habit of not hearing or a complex of not registering, manifested even when improved hearing is proven to exist.

We are grateful to Dr. L. W. Alexander for his permission to report Case 2.

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XX.

THE MANAGEMENT OF MALIGNANCIES OF THE SINUSES.*

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The history of cancer of the sinuses is familiar. It was extant before the oldest medical records, and early treatment was futile. That courageous English surgeon who some three hundred years ago treated his king with an actual cautery for what was probably a cancer of the antrum undoubtedly carried out one of the first scientific efforts to combat this disease. Other sporadic efforts are recorded, but the scientific treatment of cancer is recent. With the development of modern surgical methods, extirpation became possible and was used with and without cauterization. As a life-saving measure, this was frequently adequate, but the problem of disfigurement and rehabilitation necessitated the development of plastic flaps and concealed incisions to render the cure less obnoxious. Moure, Denker and Grosgean entered the picture at this point. Later, Thomson, Tilley and others adopted, revised and modified the earlier methods and are still carrying on to the present day. The development of electrosurgery, by Clark in 1907, and the discovery and application of radium and x-ray, however, have given the surgeon of today additional means of controlling this disease.

Cautery, surgical extirpation, endothermy, x-ray and radium represent the chronologic sequence in the treatment of malignancies of the sinuses, and they were employed in any combinations that were possible at one particular time. At present all are possible, and such men as Patterson, New, Beck, Guttman, Cade, Quick, Barnes, Öhngren, Greene and others offer us the fruits of their experience in testing these various combinations. Barnes and Öhngren feel that surgical extirpation followed by radium and x-ray, with subsequent plastic operations, if necessary, have been satisfactory. Beck and Guttman do not seem enthusiastic about surgical extirpation *per se*. Patterson has obtained excellent results with endothermy and supplementary radiation. I have no information concerning the results of

*Presented before the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Portland, Maine, January 4, 1935.

New's 295 cases of malignant tumors of the upper jaw and antrum, but I believe he used diathermy and radiation. Before this, however, he has said: "The combination of cautery and radiation . . . . has produced a greater percentage of cures than previous operative measures." Douglas Quick states that from a life-saving standpoint conservative surgery for access, drainage and radiation actually gives better results than can possibly be obtained by the most extensive surgical extirpation.

No laryngologist can extricate himself from the management of malignancies. If he refuses to handle them he will probably fail in their recognition and thereby commit an error of omission.

In determining the competency of the laryngologist to handle these cases, let us consider that he is a man of average surgical ability, that his first consideration is the welfare of the patient, that he desires to manage the case in accordance with the best medical and surgical practice, that he is willing to eliminate himself at any time if by so doing the interest of the patient is better served, and, further, that he wishes to conduct his operative diagnostic procedure so that further treatment follows as a natural sequence. It should be understood, however, that he is not a radiologist.

With these prerequisites we shall investigate nine cases of malignancies of the sinuses to which the author has had to apply himself within the last three and one-half years. We shall note the methods of attack, the manner in which the cases were managed, the results obtained and the problems raised. Included in this group are sinuses both primarily and secondarily involved. Briefly, the routine in handling the cases was as follows:

(A) *Typical:*

1. X-ray (preferably with lipiodol).
2. External opening into the sinus for biopsy, window, drainage and radiation.
3. Pathologic consultation concerning the type of growth and probable prognosis.
4. Radiation (with or without further diathermy as the case required).

(B) *Atypical:*

The routine in the atypical case was extirpation, surgical or diathermy, followed by radiation to meet the demands of that particular case.

With one exception, the operative procedures were done by the author in accordance with the requirements for postoperative radiation. The radium and x-ray treatments were given by local radiologists, the State Institute for the Study of Malignant Disease in Buffalo, Douglas Quick, Johns Hopkins Hospital and the Rochester General Hospital.

The pathologic studies were important and interesting. In every case tissue was sent to at least two recognized pathologists, and in one instance pathologists in six different medical centers examined the same tissue. A general agreement as to the degree of malignancy was evident, but borderline cases brought in varied reports and different opinions concerning treatment and prognosis. Case No. 8 illustrates this point.

CHART I.—CASE 8.

Pathologist	*Diagnosis*	*Suggested Treatment*	*Prognosis*
J. Ewing, New York	Glandular type of growth, radiosensitive	Radiation	Good
C. V. Weller, Ann Arbor	Round cell carcinoma	Surgical extirpation	Guarded
I. Gaspar, Rochester, N. Y.	Angiosarcoma	Radiation	Guarded
G. H. Whipple, Rochester, N. Y.	Highly malignant tumor, radiosensitive	Radiation	Fair
B. T. Simpson, Buffalo	Epithelioma	Radiation	Good

The results indicate that the assumption of Dr. Ewing and others was correct. Broders' method of grading cancer appeals to me as a surgeon, but I could get neither Ewing nor Weller to commit themselves to any fixed formula for treatment or prognostication. The most important responsibility of the pathologist appears to be in the recognition of radiosensitive types.

Chart II gives a summary of the facts concerning these cases.

The present status of these nine cases after three and a half years is as follows:

Five are dead. One is not expected to live. One (Case No. 7, the giant cell sarcoma) is slow growing and continues to progress. Two cases (Cases Nos. 8 and 9) are presented as cures.

CHART II.

Case	Diagnosis	Involvement	Treatment	Present Status	Longevity	Complaints	Impression
1. M. B., Age 56, F.	Squamous cell carcinoma	Antrum, ethmoid, orbit	External antrum op., X-ray	Dead	9 months	Nasal discharge 6 years	Chronic maxillary sinusitis
2. W. G., Age 59, M.	Squamous cell carcinoma	Cheek, alveolar process and antrum floor	Complete extirpation, diathermy, X-ray and radium	Dead	3 years and 5 months. 10 months after operation	Sore spot in mouth	Malignancy
3. W. F., Age 63, M.	*Macroscopic carcinoma	Nasopharynx, antrum, ethmoid, orbit	X-ray (inoperable)	Dead	1 year and 2 months	Nose bleeding, deafness, pain	Anemia, malignancy
4. L. B., Age 65, M.	Round cell sarcoma	Frontal orbit and general metastasis	External frontal operation	Dead	7 months	Pain in back, bulging eye	Mucocele, question of malignancy
5. W. P., Age 73, M.	Infiltrating squamous cell carcinoma	Antrum, cheek, orbit, malar region	Complete extirpation, diathermy	Dead	1 day	Fistula in cheek	Malignancy
6. A. E., Age 53, F.	Trans. cell carcinoma	Antrum	External antrum op., X-ray and radium	Alive	1 year plus	Pain	Malignancy
7. B. V., Age 29, F.	Giant cell sarcoma	Jaw, antrum floor, mandible	X-ray	Alive	8 years	Loosening of teeth	Malignancy
8. H. G., Age 42, F.	Radio-sensitive epithelioma	Antrum and orbit	External antrum op., X-ray and radium	Alive	2 years and 6 mos. plus	Pain	Malignancy
9. M. G., Age 24, F.	Osteoma	Antrum	External antrum operation	Alive	3 years and 4 mos. plus	Pain	Chronic maxillary sinusitis

*Specimen unsatisfactory.

Incidental information:

In two cases, or 22 per cent, the diagnosis was unsuspected.
Six cases were far advanced at the time of the first examination.
One case died as a direct result of the operation.
Four died as a direct result of the malignancy.

One highly malignant case is presented as a cure (Case No. 8, radiosensitive).

Two cases (the osteoma and the giant cell sarcoma) are obviously not malignant in the same sense as the others.

Percentage rates of success or failure may be so varied as to be almost valueless. After three and a half years the percentage of successful results was 45 per cent. If, however, we eliminate the osteoma and giant cell sarcoma, it is 16 per cent.

CONCLUSIONS.

1. Too many of these patients died. If it had been permitted, should more radical extirpation have been done?

2. Primary neoplasms of the sinuses are usually seen late because the symptoms appear late.

3. Early operation for biopsy, access and drainage is indicated for diagnosis and may be sufficient to render further surgery unnecessary.

4. Pathologic consultation is necessary for the proper conduct of the case.

5. Subsequent surgery or diathermy should be employed only after special study.

6. Radiation should be relied upon as much as possible.

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XXI.

RETROPHARYNGEAL ABSCESS DUE TO EXTERNAL INJURY OF THE NECK.*

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Retropharyngeal abscess, which is quite common in the early years, seldom can be seen in adult patients. The reason for this is the special anatomic distribution of the lymph glands of the neck.[1] There are permanent lymph glands which are located before the cervical vertebra on both sides, and there are small lymph glands which can be found on the middle line of the newborn in its first few years, but later on they disappear. This small gland is involved in cases of small children and gives the symptoms of the characteristics of retropharyngeal abscess. The formation of a retropharyngeal abscess may be caused by:

First. Upper respiratory infections (grippe, scarlet fever, measles, etc.). These cases are usually benign in character and after proper treatment they clear up in a short time. They usually develop in the first two years of childhood.

Second. In the later age, injuries of the posterior pharyngeal wall. (Foreign body, fishbone or any other hard objects, esophagoscope.) The injury which develops from these causes usually involves the superficial structures in the form of acute cellulitis and spreads down in the loose connective tissue sheets to the mediastinum or to the pleura and causes septic mediastinitis or septic pleuritis. These conditions are usually lethal.

Third. Tuberculosis which may lead to abscess formation of the posterior pharyngeal wall. Tuberculosis of the upper cervical vertebræ causes a breaking down of the bony tissue, and the abscess descends and forms the so-called cold abscess. The history and the clinical findings of Pott's disease will give the right diagnosis.

Fourth. Chronic otitis media. Dan McKenzie[2] reports about sixty cases from the literature. Urbantschitsch[3] made a thorough study of the development of these conditions.

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*From the nose and throat department of the Harlem Eye and Ear Hospital and the Post Graduate Hospital of the Columbia University.

Fifth. Infected sinuses, especially involvement of the sphenoid, may cause a retropharyngeal abscess as in the case of Grunwald.[1]

In the case which I am reporting none of the causes could be found and the unusual history and the most complicated causes warrants a thorough description.

REPORT OF CASE.

CASE 1.—G. L., 57 years of age, carpenter, came to the Harlem Eye and Ear Hospital clinic on January 16, 1934, with the following complaints:

Difficulty in swallowing, purulent discharge, with foul odor from the mouth and much pain at any movement of the head, particularly sideways.

At this time he gave a history that he contracted a cold on the 27th of December 1933, while he was working, and a few days later he developed a sore throat and pain in the neck. Later on he noticed the purulent sputum and the foul odor. A gargle did not improve his condition, which became worse. A few days later he decided to visit our clinic, where he presented the following picture:

The patient was a well built male who had lost fifteen pounds since his sickness. The family history—twice married; had five children, with no history of miscarriages; father died of diabetes at age of 54 years; mother died of diabetes at age of 74 years; sister and brother also have diabetes. He himself had no knowledge of diabetes. Nose and throat examination practically negative. Tonsils small; buried; infected. On the posterior pharyngeal wall, right behind and partly covered with the uvula in the middle line, there was an ulceration, 1 cm. in diameter, covered with grayish material. The soft tissue all around was swollen but not inflamed. Upon pressure of the soft tissue, thick yellow pus and a tenacious gray slough came out from the ulcer, the edges of which were covered with granulation tissue. The slough could be removed and showed that it had some deep connection with the fascia of the spine. The removed slough had a very offensive odor. There was a large sinus in the midline which led to a rough, hard surface. There was considerable pain in moving the head in different directions, particularly sidewise, where the motion was very limited. The throat itself was not painful, neither congested nor inflamed. (Fig. 1.) My first thought was of a cold abscess due to tuberculosis of the spine, but I could not find any definite history or symptoms to prove this. I advised the patient to have x-rays of the spine made and to have a general physical examination.

The Wassermann test was negative; smear from the ulcer showed Vincent's organisms. Culture from the ulcer showed a gram positive coccus of a saprophitic type, alpha streptococci predominating. Few pus cells, very many fusiform bacilli and spirilliform bacilli and few streptococci. Blood examination, 90 per cent hem., 4,800,000 R. B. C., 10,500 W. B. C., with 64 per cent polys and 4 per cent eosinophiles.

Due to the above findings, the patient was treated with irrigations, neosalvarsan locally and other remedies for Vincent's without any improvement. Due to the offensive odor and the persistence of the ulceration and the granulations, malignancy was suspected and a biopsy was made. Report (Dr. Meeker, Post Graduate Hospital, January 27, 1934): "Ulcerations with granulation tissue and associated hyperplasia of the epithelial covering, apparently denoting a healing in process. The sections examined do not show malignant changes."

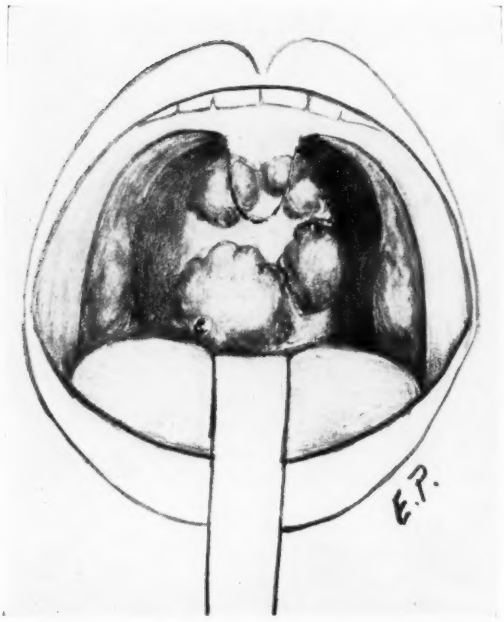

Fig. 1. Showing the ulceration on the posterior pharyngeal wall.

Examination of the urine showed: Specific gravity 1030, traces of albumen, few hyalin casts and 3.3 per cent sugar. Meanwhile some of the slough was removed; the remains were cauterized with strong silver solution; the odor diminished and the pain in the neck also lessened. The patient was advised to take care of the diabetes and was referred to the medical department of the Post Graduate Hospital and at the same time decided to take another set of x-rays.

The x-ray report, February 1, 1934 (Post Graduate Hospital, Dr. Meyer): "Examination shows an extensive soft tissue infiltration in the retropharyngeal area, extending practically the full length of the cervical region in front of the spine. Irregular calcareous deposits are observed just back of the thyroid cartilage, the picture suggesting inflammatory soft tissue infiltration and probable abscess rather than of neoplasm. Slight osteo-arthritic changes observed in the articular border of the vertebræ in the midcervical region." These are not infrequent at this age and perhaps without any definite connection with the soft tissue infiltrative process. (Fig. 2.)

February 14, 1934, the urine showed: specific gravity 1036, traces of albumen without casts, sugar 3.3 per cent; blood sugar, 0.285.

On one occasion the patient, observing that no diagnosis had been made, changed his history of the onset and told us that on the 27th of December, 1933, while working he hit his neck on a beam and although he did not have any complaint at this time, he started to have pain in the neck a few days later; still the injury of the spine could not be substantiated by the two previous negative x-ray findings.

Fig. 2. Showing the abnormal thickness of the soft tissues on the postpharyngeal wall.

The patient's condition remained the same until March 21st, and in the meantime he received insulin and suitable diet at the Post Graduate Hospital Medical Clinic and was treated locally by me two and three times a week. On the above date he came to the clinic complaining of extreme weakness, headache, loss of appetite and very much pain in the neck. He gave the impression of being a very sick man. Upon examination, his throat condition was about the same, but there was a distinct acetone odor from his breath, and although the urine was negative for sugar and acetone he was referred to the Medical Department with the thought that his condition was turning worse due to his diabetic condition. On the 23rd he was admitted to the Post Graduate Hospital, where the routine examinations were done. He was put on diet and insulin. On March 24, 25 and 26 his condition was about the same. He was fairly comfortable walking around, but had pain, headaches and was restless, particularly at night. Temperature on the 26th was 100.4. March 27th the temperature was 101.4. The Mantoux test for tuberculosis was negative. An orthopedic surgeon was called. On March 28th another deep biopsy, another x-ray and lumbar puncture for Wassermann and for culture were taken. At this time the x-ray (Dr. Meyer) showed: "Soft tissue infiltration about the same. Further evidence of osseous involvement exists insofar as the anterior margins of the bodies of

the second and third cervical vertebrae are eroded with either débris or secondary reactive changes in the overlying soft tissues." The biopsy report by Dr. Meeker disclosed "Polypoid hyperplasia with superficial ulceration and granulation tissue formation with purulent exudate. Negative for neoplasm." The report on the spinal puncture: No increased pressure, clear, white cells, 65 per cmm., mononucl. 85 to 95 per cent, lymph 10 to 15 per cent. On March 29th, temperature 102, very much pain in the back of the neck. Mind occasionally confused, probably following the puncture. The orthopedic surgeon who was called for consultation did not succeed in examining him due to his condition. On March 30 and 31, his condition was better again. Temperature went down to 99, and he was walking around. He complained of much pain in the back of the neck all the time. On the morning of April 1st he suddenly had a chill, his temperature went up to 104 and he became irrational and unconscious at times. Retention of urine (catheterized), some stiffness of the neck and headache. Medical consultations and orthopedic consultations were held. Medical examination: The lungs are clear throughout; the heart is negative except for soft mitral systolic blow, apparently functional and not transmitted. Abdomen negative, so far as can be determined, with complete lack of cooperation. No signs of meningitis. Pupils react to light. Deep reflexes are all diminished. Kernig and Babinski signs negative. No clonus. Patient appears very toxic and apparently the signs are coming from this. The cells in spinal fluid may be explained on basis of meningismus. The diabetes appears to be controlled, judging from the urine, which is negative under insulin, 18-5-15; this, however, should be watched closely, especially in the face of the severe infection. Another blood sugar estimation should be made.

On April 2, 1934, patient acutely ill, extremely toxic, easily roused and responds rationally. Complains of pain and stiffness of the neck. Unable to turn head or bend forward because of associated pain. In the lungs there is evidence of acute diffuse bronchitis. Rigidity of the neck still present. Mild Kernig. Spinal fluid clouded—19 white cells per cc., 840 red blood cells per cc. Polynuc. 80 per cent, lymph 20 per cent. Culture after four days negative. Blood count, red blood cells 5,420,000, leucocytes 13,650, 83 per cent polymorph, 2 per cent polymorph basoph, 15 per cent lymphocytes. Urine, specific gravity 1,020; faint traces of albumen; sugar less, 0.2 per cent; traces of acetone; no casts. The patient died around 9:30 p. m.

Autopsy report: "The posterior wall of the pharynx, opposite the second cervical vertebra, is swollen; presents greenish necrotic discoloration, and there is an ulcer in the central portion, measuring one-half inch in diameter, which extends through the necrotic anterior wall of the body of the second cervical vertebra, communicating with the space in the fractured body of the third cervical vertebra. All the tissues of the pharynx are swollen, greenish in color and the posterior wall of the pharynx is necrotic.

There is a definite sinus extending through the ulceration in the fractured body of the third cervical vertebra. The spine is opened posteriorly and shows no evidence of injury. The spinal laminæ and the cervical portions are removed. The portion of the cervical cord adjacent to the second and third cervical vertebra is definitely swollen, and on the anterior surface the dura presents a greenish, swollen appearance. The upper part of the cervical cord corresponding to these two vertebræ is distinctly swollen and softened; shows gray discoloration. On removing the cervical cord from the canal the anterior surface of the second and third cervical vertebra presents a greenish appearance, the fascia being swollen. There is a slightly thickened ridge in the fascia, extending down on the anterior surface of the spinal

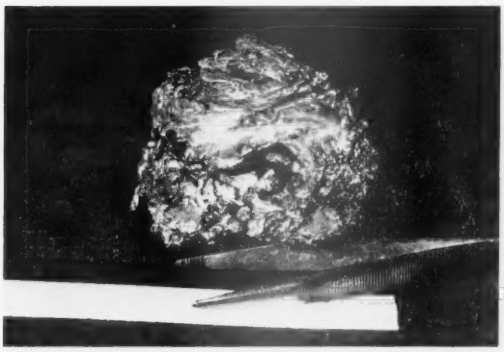

Fig. 3. Part of the spine showing a comminuted fracture of the body of the cervical vertebra.

canal and into the bodies of the fourth and fifth cervical vertebræ; this ridge is about one-fourth inch in diameter, slightly elevated; runs for a distance of about two inches. On section, this fascia is quite firm and is slightly thickened.

The first, second, third and fourth cervical vertebræ removed together and on section reveal a comminuted fracture of the body of the third cervical vertebra, the marrow being of a dark gray color. The fracture extends through the lateral surface, and there are several smaller cracks. There is a complete separation of the anterior and posterior portions of the fracture. The fractured space communicates with the sinus in the posterior pharyngeal wall, as already described. No caseous material was found within the fracture. No evidence of tumor tissue can be made out grossly.

The fracture apparently involves only the third cervical vertebra.

Anatomic diagnosis: Fracture of the body of the third cervical vertebra, with osteomyelitis. Pending microscopic examination.

Extension of the infection with necrosis of the posterior pharyngeal wall; localized. Suppurative meningitis and myelitis of the adjacent spinal cord. Lungs: Congestion, bronchopneumonia. Congestion of liver, kidneys and spleen. Heart: Coronary arteriosclerosis.

Cause of death: Fracture and osteomyelitis of the body of the third cervical vertebra. Retropharyngeal abscess. Suppurative myelitis of the cervical spinal cord. History of having been injured while at work when beam struck him on the back, on December 27, 1933."

CONCLUSION.

Without the autopsy it would have been impossible to establish a cause of the ulcer which existed more than three months on the postpharyngeal wall. Three x-rays which were taken did not show any fracture of the vertebra which can readily be recognized on the part of the spine obtained at the autopsy. The failure of the x-ray in certain spinal injuries is well known among the orthopedic surgeons

and radiologists. As to the diagnosis of the injury itself I have to quote Kleinfeld,[5] who said that among the industrial injuries the fracture of the spine is quite frequent but recognition at times is very difficult. These difficulties arise from one or more of the three very distinct causes. First, the lack of appreciation of a clinical symptom-complex which indicates the existence of the injury of the spine. Second, the symptoms, both objective and subjective, may be so mild that they are readily explained by some simple injury such as contusion or sprain. In fact, they may be so mild that the patient does not pay any attention to them until they have lasted for many weeks or even months. Third, some one symptom may be so prominent as to distract attention from the spine.

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XXII.

EXTERNAL DEFORMITIES CORRECTED AND REMOVAL OF EXISTING INTRANASAL OBSTRUCTION ADVANTAGEOUSLY ACCOMPLISHED AT SAME OPERATION.

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It is a well known fact that many persons desiring correction of external nasal deformities also suffer from more or less marked difficulty in breathing, due to co-existing intranasal obstruction. All rhinologists interested in rhinoplasty agree, therefore, that external deformities should not be corrected while the intranasal obstruction is permitted to remain. Such harmful obstruction should be rectified, not only to furnish comfortable breathing but also because of its ill effects upon general health. Of course, the younger the subject the more pronounced are such ill effects.

For many years it was our custom first to correct intranasal defects by an operation performed six to twelve weeks prior to that done for the external deformity. This method of procedure we found to be a distinct hardship to the patient, not only because he must submit to two operations instead of one, but also because of the increased expense entailed by double hospitalization and added loss of time. In not a few instances objection to two definite operative procedures has been so great as to cause the patient to say, "Oh, just correct the external deformity and leave the inside alone." In a few of our earlier cases, where we were persuaded to do this, it was to our great regret, because of complaints from patients later of inability to breathe properly and their insistence that something be again done for the relief of the discomfort.

For the past three years, therefore, we have been correcting the intranasal condition and the external deformity at the same time, to the entire satisfaction of the patient and ourselves. Besides we find it easier to lower the dorsal plane, when necessary, or correct a lateral deviation of the nose, if a modified submucous resection is done at the same sitting. Only when a rib graft is required in the correction

of a saddle nose would we advise against the dual procedure, owing to a greater likelihood of infecting the graft through added traumatization of the tissues. In doing a rib graft it is our fixed rule, after preparing the nose for its reception, to obtain the graft from the rib to shape, and place it in position under the skin of the nose as quickly as possible, with the least trauma to the nasal tissues. By following this rule we have not experienced a single infection of our grafts. In saddle nose, therefore, any necessary intranasal surgery is done at least six weeks prior to the plastic operation.

Of course, it goes without saying that no plastic surgery should be attempted in the presence of a purulent sinusitis. We must be certain first that such condition has been entirely cured.

In certain types of deformity, such as, for instance, the scoliotic nose, which invariably follows fracture of the nasal bones, we generally find one or both sides of the nose markedly obstructed by septal deflection resulting from fracture and displacement of the septum at the time of the injury. Many of these patients assure us that, whereas prior to the accident they rarely had colds, since that time they have been victims of frequently recurring and tenacious intranasal infections, often accompanied by sinus involvement. In some such conditions of long standing, as a result of these repeated colds, we find also marked hypertrophy of the lower turbinate bodies, with large pendulous posterior ends. These posterior ends not only often completely block the passage of air through the posterior nares, but, by increasing the flow of mucus into the nasopharynx, bring about frequent upper respiratory infections and at times actual disturbance of the gastro-intestinal tract. Besides, by encroaching upon the lumen of the eustachian orifices, interference with the proper ventilation of the middle ear occurs, causing frequent catarrhal exudates as well as purulent otitis media, with or without complications—a well known danger to the hearing, even to life itself.

In not a few of the long acquiline humped nose cases, we have found a pair of bullous middle turbinates, so large at times as to make constant pressure contact against the septum. Such patients usually complain not only of an almost constant sensation of pressure about the root of the nose and frequently recurring headaches, but many also complain of prolonged attacks of sneezing, accompanied by profuse and annoying hydrorrhea resembling allergic attacks. From such attacks we have seen complete relief follow excision of these bullous middle turbinates in individuals who had been otherwise treated without results.

Although I am fully aware of the almost universally prevailing objection to the removal of middle turbinates, and while heartily agreeing that no needless sacrifice of nasal tissue should ever be made, yet at the risk of having much criticism heaped upon me, I cannot refrain from expressing a conviction, bred of long experience, that many failures to obtain functional results, relief from reflex headaches and ample breathing space, from intranasal surgery, is due to such conservatism. Where, in doliocephalic heads with narrow nasal passages, the bullous middle turbinates make pressure contact against the septum, one, or when necessary, both of them, are completely excised by us without hesitation. This procedure has been followed by the most signal benefit to a great number of patients. Many of the cases have been followed up over a period of years, and I am certain that no ill effects have ever resulted from the loss of these middle turbinates.

In many young subjects between 16 and 18 years of age, whose difficulty followed injury to the nose in very early childhood, instead of meeting with the usual type of septal deflection, we found that, through fracture with transverse displacement of the cartilaginous septum, accompanied by marked ecchondrosis, both sides of the nasal vestibule were completely blocked. This obstruction appears as an irregular blank transverse wall of cartilage, extending from the outer limit of one vestibule to the outer limit of the other, beyond which it is impossible to see the condition of the interior of the nose. Invariably these young people have a disinclination to eat properly and appear pale and undernourished; they lose sleep because of marked interference with nasal breathing, and show poor concentration in their school or college work. Bimanual palpation over the nasal alæ reveals this type of obstruction as a hard, bulbous mass, and the usual rotundity of the cartilaginous dorsum becomes a flattened surface. It is truly amazing to observe the rapid improvement in these young people after restoration of nasal breathing.

One must not make the error, in correcting this type of obstruction, of removing too much of the obstructing cartilaginous mass, lest there be left insufficient support for the tip of the nose. It is far better to remove submucously from each side of this blank wall enough cartilage to restore sufficient breathing space, and permit the central portion to remain for the support of the nasal tip in the correction of the external deformity.

For the same reason, even in the correction of the ordinarily deflected septum, when external plastic work is contemplated, the

classic submucous resection is not a wise procedure. Much difficulty in properly supporting the tip of the nose has been encountered by us in a number of cases referred by rhinologists for plastic work, after they had previously removed too much of the triangular cartilage in the performance of a submucous resection. By doing the so-called modified resection described by me in the ANNALS of June, 1924, tip support is always assured, without sacrifice of breathing space to an appreciable degree, if at all. This is a very important point and, therefore, even though some of you may be familiar with the procedure, I am here again showing the slide and reviewing briefly the technic:

After having undermined the mucoperichondrium on one side of the septum, as is done in all septal resections, the usual vertical incision through the lower front end of the cartilage is made with the knife, and the mucoperichondrium of the opposite side is undermined. The swivel knife is now passed backward along the upper edge of the vomer until it encounters the bony portion of the septum in the rear, and is then pulled forward in the same line of incision, in order that none of the cartilage be removed. Now, with the sharp elevator, the cartilaginous septum is transfixed at its posterior attachment to the bony septum, from which it is completely severed. Thus, you will note, the triangular cartilage is left wholly attached along the bridge of the nose only. At this point, by removing the thick lower portion of the vomer along the intermaxillary ridge, with the bone forceps, space is made to allow the curved or deflected triangular cartilage to straighten out. With this accomplished, one may proceed with the operation, removing as much of the deflected bony portion of the septum as may be necessary to obtain a smooth, straight septum in the rear.

In an extreme deflection, where obtaining a straight septum seems almost impossible without removing practically the whole of the bony septum, another word of caution is offered, lest through removal of too much bone we later have a flail membranous septum without bony support, which flips to one side on inspiration—impairing the ingress of air through that side—and flops to the other side on expiration, impairing the egress through the opposite side. I know of nothing in the nose more difficult to correct than this type of flail membranous septum, or nothing which causes the individual more annoyance through its marked interference with nasal breathing.

To prevent such an occurrence, only the thickest portion of the bony septum should be removed. Then, with the Struyken scissors,

the thin upper portion, which is made up of the perpendicular plate of the ethmoid, is severed from its attachment to the base of the skull, so that it may be dropped down into the pocket between the septal membranes. This transplanting of the perpendicular plate to the lower portion of the septal pocket absolutely precludes the occurrence of the flail septum, and at the same time, through thinning of the upper half of the partition, makes for greater space in the upper narrowest portion of each side of the nose. Of course, the intermaxillary ridge may be removed with the hammer and chisel, as is usually done, but as much of the vomer bone as possible should always be retained, even if severed from its attachments, to aid in supporting the septal membranes.

So far as hypertrophied lower turbinated bodies are concerned, we endeavor to excise as little of the soft tissue as possible, and rarely if ever any of the bone itself. We prefer infracting the bones and

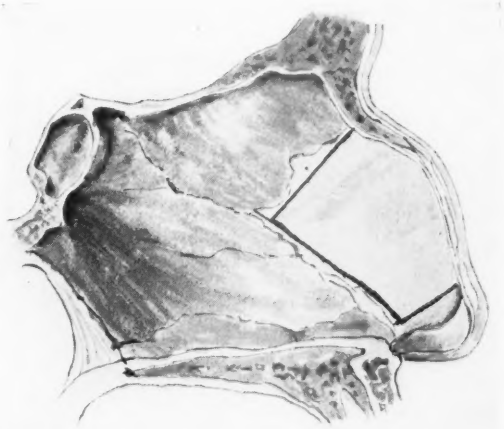


Fig. 1. Schematic drawing indicating incision in triangular cartilage, used in *modified* submucous resection.

forcibly pressing them outward against the antral walls, removing only the enlarged pendulous posterior ends with the snare. This furnishes sufficient breathing space in the vast majority of cases, but should it occasionally not do so, it is an easy matter a few weeks later, as an office procedure, to reduce the still swollen turbinate body, either by galvanocauterization or by the use of the high frequency current.

We have performed the dual operation in thirty or more cases, with uniformly satisfactory functional and cosmetic results. Time in

the hospital averages five days—the same as when the external deformity alone is corrected—while postoperative care in the office is not prolonged beyond that accorded a simple rhinoplastic case.

Our technic can best be made clear by describing in detail a few of our typical cases:

REPORT OF CASES.

I. F. R., age 29, first consulted us September 22, 1934. About seven years prior she was in an automobile accident, sustaining an injury to the nose. Examination showed a convex vertical deformity, fairly well marked hump on the dorsum, and a rather over-sized cartilaginous nose with drooping tip. Directly to the left of center of tip was a scar about one-half inch long, caused by a cut from the broken wind shield. Internally we found a rather marked obstruction, due to deflection of septum to the right, along with prominent hypertrophied and intumescent lower turbinates with large pendulous posterior ends. Patient complained of great difficulty in breathing and was highly conscious of the deformity.

On November 19, 1934, under local anesthesia, augmented by three capsules of three grains each sodium amytal, a v-shaped section was removed from the subseptum. The base of this section, about 10 mm. in width, lay at the dorsum nasi just above the tip and the apex at the anterior nasal spine; through the defect thus created, the membrane on each side of the septum was undermined, and a modified submucous resection performed. Double lower turbinate compression followed, and both enlarged posterior lower turbinate ends were removed with snare. This having been accomplished, both nasal cavities were lightly packed with sterile nu-gauze tape, before proceeding with the correction of the external deformity.

A bilateral incision within each vestibule at the junction of each ala with the bony nose was now made, and joined over the top of the septum nasi. These incisions included the lining of the vestibule and overlying cartilage up to the skin covering the bony nose, which was undermined in its entirety. The irregularities and hump on the bony dorsum were now removed with rasp. Closing the subseptal defect with four black silk sutures on each side elevated the tip and shortened the nose.

To shorten the sides of the nose, v-shaped sections were now cut from the upper edge of each ala incision, to the extent that they overlapped the lower edges, but left attached at their bases where they join with the mucous membrane of the septum. The apices of these attached triangular flaps, together with one-third of their length, were cut away and the remaining two-thirds turned upward over the septum of the nose, with their raw surfaces apposing, and fastened in this position with one black silk mattress suture. These flaps so placed served to obliterate an existing depression of the dorsum just above the tip, which otherwise would have marred our result.

After removing the sterile tape temporarily placed, the interior of the nose was again packed loosely with one-half inch iodoform nu-gauze tape, between the septum and middle turbinates. Two soft cigarette packs on each side to support the septum (as we do in regular submucous operations) were now placed in the lower portion of each nasal cavity and the vestibule filled with iodoform tape, the lower ends of which were carried over the columella from one vestibule to the other three or four times, to protect the subseptal wound. The copper saddle splint was now applied in the usual way.

Case 1.

Case 2.

Forty-eight hours later all packs were removed, but the vestibule was again loosely packed as before with iodoform tape. On the fifth day all packs were left out of the nose, and on the seventh the splint was removed, the skin bathed with 95 per cent alcohol, and after removing all intranasal sutures the splint was again applied. From now on the splint was changed every fourth day (in order to take care of the skin) for three weeks, when it was discontinued.

2. Milton S., age 20 years, was seen by us October 31, 1933, giving the following history: At the age of eight, while riding on the rear seat of a motorcycle, he

Case 3.

Case 4. Rib graft.

fell and fractured his nose. After swelling subsided, the nose was seen to be very crooked and displaced toward the right side. For the past five or six years he experienced great difficulty in breathing through the nose, was annoyed by frequent attacks of sneezing and more or less continuous postnasal dripping. He also suffered often from severe headaches.

The external nose was of the scoliotic type, being displaced also toward the right cheek, and the bony dorsum was made irregular by exostoses along the line of fracture. Internally the nose was greatly obstructed by an S-shaped septal deflection,

the cartilaginous portion curving greatly toward the left, while the bony portion bent toward the right side. On the summit of the bony deflection there was a well marked ledge where the vomer and perpendicular plate of the ethmoid joined. Markedly hypertrophied lower turbinates, along with a large bullous left middle turbinate, served to make almost a complete obstruction.

On November 1, 1933, at Sinai Hospital, patient was operated upon under local anesthesia, augmented by sodium amytal and twilight sleep.

A modified submucous resection was first performed through the usual incision, the septum straightened and the thickened ledge removed. Compression of both lower turbinates followed, forcing them outward against the antral walls, and removal of the posterior pendulous ends with the snare. Now the large bullous left middle turbinate was completely excised and the nasal cavity packed loosely with nu-gauze tape, preparatory to correcting the external deformity.

The usual bilateral intranasal incisions were now made along the junction of each ala with the bony nose, and through these the skin over the entire bony dorsum was undermined. All irregularities of the dorsum were smoothed with the rasp, before completely mobilizing the nose, by severing with the saw the nasal bones at their attachment to the nasal processes and infracting their attachment to the frontal bone with hammer and buffer, and Carters' bone forceps.

The nose was thus rendered so flail that it could be placed in any desired position toward the midline of the face, without any tendency to spontaneous displacement. Temporary packing was now removed, and the usual iodoform tape and cigarette packing inserted, after which the splint was applied.

This patient returned to his home in Pittsburgh after four weeks with a very happy cosmetic and functional result.

I should like also to give you just a glimpse of a few other cases wherein the dual procedure was successfully carried out:

(Cases shown.)

In closing, I would emphasize that from our experience where plastic work is required, the welfare of the patient is best served when the plastic operation for external deformities and the necessary correction of intranasal obstructive conditions are considered as a joint procedure.

1820 EUTAW PLACE.

XXIII.

BRUCELLOSIS AND OTOLARYNGOLOGY.

CAESAR HIRSCH, M. D.,

NEW YORK.

It is more than twenty-five years since the Danish veterinary Bang discovered the causal organism of the contagious abortion of cattle. But only in 1922 was the first case of human infection with the abortus variety of the organism diagnosed (Johns Hopkins Hospital) and reported by Keefer[1] in 1924.

In Middle and Northern Europe it was not until 1927 that the attention of physicians was called to the so-called Bang disease. Subsequently, as physicians gradually learned that brucellosis is a disease that should be considered in every case of fever not attributable to other causes, the number of reported cases in this country has greatly increased.

In 1933 there were 1987 cases reported in the United States. A. C. Evans[2] states that she is certain that a great number of cases of brucella infection are not even recognized as such.

While differentiation between the brucella melitensis and Bang's bacillus with abortion serum is not possible, serum of patients suffering from Mediterranean fever agglutinates maltese races at a higher titer than races of abortus. Therefore, the germs are supposed to be very closely related, though not identical. This was also stated by A. C. Evans, who in 1917 studied the bacterial flora of milk as it comes from the cow's udder. Later on, the name of the discoverer of brucella (Bruce) was given to the entire group, including the organisms of Bruce and Bang, and so in this country the disease is now called "brucellosis."

It must be mentioned, however, that even though Bang's bacillus and Bruce's micrococcus are alike morphologically, they vary epidemiologically insofar as the germ, causing the undulant fever, has a much higher pathogenesis for man than Bang's bacillus. Being of the opinion that some otolaryngologists have not as yet dealt with this important illness, I beg permission to speak about it briefly.

The infection may take the peroral, i. e., intestinal or percutaneous way. Milk or urine as well as aborted secretions may contain the

Bang bacillus. It has not yet been established which mode of infection is more frequent, but probably most of the cases are caused by drinking raw milk and only very few cases are due to contact infection. Among the latter cases we find especially farmers, butchers and veterinarians. Infection from man to man is very rare. As compared to women, men fall victims to the disease at a ratio of 3:1 and usually between the fifteenth and fortieth years. Children seem to be more immune than adults. While all other investigators have emphasized that they did not know of any case under eight years of age, Kristensen and Holm[5] report 500 cases of brucellosis without a single case under five years and only five cases between five and ten years of age. Hardy[1] and his collaborators have found among their 354 cases, four cases in children under five, and eight cases in children between five and ten years of age. This is astonishing, since children drink milk more frequently than adults and are certainly more exposed to infection through dirt.

In 1922, before human infection with the bovine type of organism was known to occur, Basset-Smith[3] made the statement that in its early stages nearly every case of brucellosis had been treated for some other condition before a correct diagnosis was made. Every treatise on brucellosis emphasizes the extreme difficulty of making a diagnosis, owing to the lack of characteristic symptoms. Typhoid, malaria, rheumatic fever, endocarditis, tuberculosis, broncho-pneumonia, bronchitis and influenza are some of the erroneous diagnoses made of acute cases of brucellosis. There are also mild types of brucellosis and perhaps these are the cases which the otologists are confronted with more often than the fulminant cases running high temperatures.

Shaw[6] was the first to recognize the so-called ambulant form of the disease in which the symptoms were entirely absent or limited to a few days of slight fever. Shaw discovered the ambulant type by examining the blood of 525 Maltese dockyard employees for specific agglutinins in the blood serum. Fifteen per cent gave a distinct reaction and twenty-two of those giving the highest agglutinin titer were examined carefully. Cultures were obtained from ten: all of these patients were working full time. Examination revealed, however, that some of them showed a slight rise of temperature.

Hardy, Jordan, Borts and Campbell[1] confirmed Shaw's observation, and the only constant symptom they found in their patients was weakness. The chronic cases, of course, do not very often show a rise of temperature and very seldom a temperature so high that the name "undulant fever" could be used.

Hughes[7] describes the so-called intermittent type of the fever as cases with a temperature of no more than 99° F. in the afternoon and a normal or even subnormal temperature in the morning or at night.

Feldman and Olson[8] studied the chronic disease in the swine. They selected apparently healthy animals that gave positive agglutinin reactions. From one of these animals, in which postmortem examination revealed no lesions, the causal organism was obtained from the spleen. These investigators also studied a number of cases of spondylitis in apparently normal slaughter house hogs. They found the brucella usually in the lumbar or sacral vertebrae. This finding is particularly interesting, because in the human disease lumbar pain with objective signs of the disease is a known symptom in cases with brucella infection.

In 1929, when I[9] published two case reports of brucellosis with pharyngitis, I drew the attention of otologists to the fact that cases with ear affections would probably also be observed.

At the convention of the Danish Otological Society, in the year 1932, Hvidt[10] reported the case of a 30-year-old man suffering from brucellosis and a bilateral impairment of hearing for high pitched tones and slight changes in the spinal fluid.

Bergmark,[11] in 1933, reported a case of a 55-year-old man suffering from a meningo-encephalitis caused by "bacillus aborti Bang." The patient was examined otologically by Bárány, who stated an impaired hearing of central origin was present. Postmortem examination showed a definite infiltration with round cells in the pia mater, especially the region of the Sylvian fissure and around the vessels of the brain substance. Pathology of the auditory nerves and labyrinths was not further gone into.

The first detailed description of a case of ear affection in brucellosis we owe to Videbech[12]:

> A 20-year-old girl, a packing house employee, was admitted to the neurologic department of the Kommune Hospital in Copenhagen, August 31, 1932. Her illness began July, 1932, with nausea, vomiting, pains in abdomen, lumbago, neuralgiform pains and weakness of the lower extremities. There was a beginning paralysis of both legs with loss of patellar reflex and Babinski's sign bilaterally positive. X-ray of vertebral spine showed no pathology. Vidal's reaction was positive for Bang's bacillus 1:400 in the blood, as well as in the spinal fluid. Definite complement fixation for Bang. Temperature 38 to 38.6° C.
>
> Rising temperature was accompanied by slight pains in the pharynx. Treatment with typhoid vaccine. There was a gradual diminution of paresis of the legs and the patient was able to walk again.

Early in 1933 the patient complained of pain in both ears and the entire head. Otoscopy was normal but both mastoid bones were sensitive to pressure. Impairment of hearing occurred on both sides with periodic tinnitus and slight vertigo. She had never had trouble with her ears prior to this. No medication was used which might have caused ear symptoms. February 4, 1933, patient was examined at the otological department of the Kommune Hospital. The ear drums were slightly muddy, but no retraction or any other changes were observed.

Hearing tests:

Whispered voice both sides 2.5 meters; conversation, both sides 20 meters.

a_1 Air 38″ (55″) a_1 M 11″ (15″) L.i. 17″

38″ 6″ 18″

L.s. 15,500 (monochord)

14,500

Romberg's test was not possible on account of paresis of the lower extremities. No spontaneous nystagmus. Rotation test (after 5 rotations):

rotation to the right ———> 11 seconds (3 oscillations)
rotation to the left <——— 11 seconds (3 oscillations)

March 13th: L.s. 15,000 L.i. 16

14,000 16

Spontaneous nystagmus ———> r. and l. slight.
Caloric test 22 cc. 18° (C) r. ear ———> 40 seconds.

Change of direction after bending head forward:

Left ear <——— 20 seconds and change of direction.

No spontaneous past pointing. No dysdiadokokinesis.

Obviously this is a case with distinct impairment of the high pitched tones and slight anomalies of the vestibular apparatus. There were no symptoms of an affection of cerebral nerves, except trifacial symptoms. Affection is bilateral and equally pronounced on both sides; that there is a lesion of the nuclei is, therefore, very improbable. Low pitched tones being intact, seat of the trouble in the labyrinth is not probable. The most natural hypothesis would point to bilateral neuritis acustica. This type of neuritis is very frequently encountered in acute infectious diseases, especially typhoid fever.

P. Cantaloube[13] found in undulant fever, which is so closely related to Bang's disease, that in 20 per cent of all cases, especially advanced cases, suffered from impaired hearing. Normal hearing was usually regained over a certain period. Cantaloube, however, also mentions two cases of complete one-sided deafness which developed after two months without pain or tinnitus. Sometimes noc-

turnal otalgias have been observed. Only in one case was otitis media observed. E. Saquépée[14] mentions temporary impairment of hearing and sometimes deafness in cases of undulant fever. Roger, L. Rimbaud and M. Janbon[15] describe a "syndrome encéphalomeningé de la neuromelitococcie." Roger reports five cases of subacute meningitis and impaired hearing which developed several months or years following undulant fever. Rimbaud et Janbon[15] report two analogous cases with the following symptoms:

1. Paroxystic manifestations: paresthesias in the upper extremities, in tongue and face, migraine and sometimes disturbance of speech.

2. Permanent symptoms: Fatigue, weakness of memory and labyrinth trouble, vertigo, tinnitus and impaired hearing; in both cases there were changes in the spinal fluid.

Are these cases etiologically caused through Bang's disease? In none of these cases was there a question of a minor middle ear affection. At any rate, it is imperative to pay attention to the ear in brucellosis.

Besides the above mentioned cases of brucellosis involving the ear, I reported two cases of pharyngitis in brucellosis in 1929. Lieberherr[16] reports that he found pharyngitis and laryngitis present in eleven out of his twenty-four cases. In how many of these eleven cases pharyngitis and laryngitis appeared separately he does not say. The following case may add to the knowledge of involvement of the ear in brucellosis.

REPORT OF A CASE.

CASE 1.—The 32-year-old farmer's daughter, A. P., came to me May 15, 1932, complaining of bilateral tinnitus. The patient stated that she had been perfectly well all her life and that there was no case of defective hearing in her family. Three months ago she suffered from an illness with high fever and was treated for typhoid fever. Later she was told that she was suffering from Bang infection. The attending physician wrote that the patient was taken ill suddenly with high fever and chills, vomiting and severe headaches and vertigo. Her temperature rose as high as 105° F., showing distinct remissions. Patient was also suffering with a severe bronchitis.

Blood Count: 3,700 white blood corpuscles, 48 per cent neutrophiles, 42 per cent lymphocytes, 5 per cent monocytes and 5 per cent eosinophiles, red blood corpuscles 4,3 millions and 70 per cent hemoglobin. All findings together with an enlarged spleen pointed to typhoid fever. Bacteriologic and serologic examinations, however, showed neither typhoid nor paratyphoid reactions.

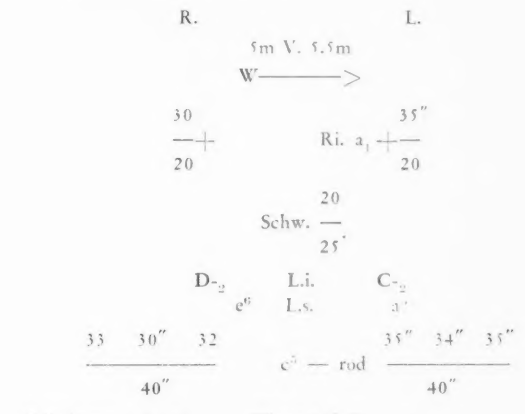
The acute severe state of the patient, who complained intensely of muscular pains, pains in joints and back during this time, improved after several weeks. The attacks of high fever were recurrent, however. In the meantime the physician

learned that other people, living in the same district, suffered from similar illnesses and that several cases of infectious abortion had occurred amongst the cattle. He, therefore, sent the blood of the patient for serum agglutination test and found that it agglutinated very distinctly in a titer of 1 to 1,600.

The treatment consisted of small doses of aspirin and quinin. As soon as the diagnosis showed "Bang," he administered three intravenous injections of trypaflavine (5 cc. of the 2 per cent solution). After two months the patient was able to return to her work, although her temperature was frequently still above 100°F. and slight pains in the back remained. But the patient failed to take her temperature any more. According to the physician's memory, patient had complained of bilateral tinnitus and vertigo very early in the disease which symptoms he blamed on the high temperature.

Findings on May 15, 1932:

Both ear drums normal otoscopically; no spontaneous nystagmus; no spontaneous past pointing. Romberg negative.



Caloric test: 5 ccm Water 13 C.

R. 10″ latent period 205″ nystagmus period.

Pastpointing+-+ Fall+-+ Vertigo+-+

L. 12″ latent period 195″ nystagmus period.

Fall+ Vertigo+-+

Past Pointing+

According to these findings, we were dealing with a bilateral disturbance of the inner ear, more pronounced on the right than on the left side. But obviously it was not only the cochlear nerve, but also both vestibular branches which were affected. Even though there was no spontaneous nystagmus, the short period of latency showed a distinct "readiness" for nystagmus.

The long duration of nystagmus and the strong subjective and objective reactions pointed to a hyperirritability of both vestibular nerves ($r > 1$). We requested the patient to take her temperature again with the result that her eve-

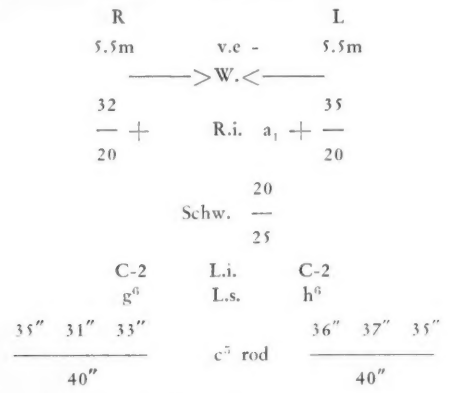
ning temperature frequently rose to 101.2°F. Blood count showed 5900 white blood cells; 38.7 per cent leucocytes; 15.3 per cent staff cells; 7 per cent monocytes; 39 per cent lymphocytes; 0 eosinophiles; 0 basophiles. The serum of patient agglutinated Bang in the titer 1:1000.

The patient received intramuscular injections of antimelitococcic serum for ten days, i. e., the first day 20 cc., the second and third days 30 cc. each, and after a lapse of three days 40 cc. for three successive days.

Supposing that we were dealing with a circumscribed meningitis similar to the case of Roger, Rimbaud and Janbon; an intravenous injection of 5 cc of a 40 per cent urotropin solution was given daily for 14 days. Temperatures at first showed no reaction to the serum or urotropin and did not rise above 99.0°F. After the serum injection the white blood cells increased to 7500. 52.2 per cent leucocytes; 8.3 per cent staff cells; 8 per cent monocytes; 2 per cent eosinophiles; 0 basophiles; 29.5 per cent lymphocytes, i.e., approaching normality with a prognostically favorable reappearance of eosinophiles.

Subsequent report of the patient showed a marked improvement in the tinnitus and vertigo as well as diminishing pains in the back.

Functional tests of June 5th showed the following:



Caloric test: 5 cc. water of 13° (C).

R. 15″ ——> 185″ Fall-+ Vertigo++ Past Point++
L. 20 <—— 175 Fall-+ Vertigo+ Past Point+

Obviously this showed a marked improvement, although not a return to normal.

The patient went back home and no further reports were obtainable.

All symptoms showed that it was a case of bilateral acute disturbance of the cochlear and vestibular nerves. It was not probable that the disturbances of hearing and equilibrium of our patient were due to any medication. First, these symptoms appeared in the very beginning, together with the other violent symptoms, which were so stated by the physician in charge. Secondly, the doses of quinin and aspirin were, according to the statement of the physician, very

small and were taken for a short time only. As we have no post-mortem records except of the one case, in which the petrous portion of the temporal bones were not examined histologically, we can only surmise that, in cases with impairment of hearing and equilibrium due to brucellosis, we are facing a condition of the auditory nerve with degenerative changes in the nerve-ganglion apparatus. The post-mortem findings of Roger, Rimbaud and Janbon, however, permit the assumption that brucellosis, similar to parotitis epidemica, may lead to circumscribed meningitis developing into inflammations of the auditory nerve with a descending degeneration of the nerve-ganglion apparatus. Both brucellosis and parotitis show a similarity in that they both show a comparatively small mortality and orchitis.

In our case the illness still reacted to the melitococcic serum four months after its inception. A complete restitution of the function of the auditory nerve was, however, impossible, probably because the atrophic-degenerative changes could not be entirely repaired after such a long period. Had this case been immediately diagnosed as brucellosis and the serum given, a complete recovery of hearing and equilibrium might have been possible.

Carpenter and Boak[17] examined fifty-five pairs of diseased tonsils from man. The technic used by the authors was that after surgical removal each tonsil was placed in a closed sterile jar and brought to the laboratory, where it was transferred with a pair of sterile forceps to a sterile petri dish and described. Cultures were made after the surface of the tonsil had been seared with a hot spatula. Bits of the tissue or any purulent exudate were transferred to an infusion agar to which sterile horse serum had been added. The tubes were then placed in a jar in which 15 per cent of the air had been replaced with 10 per cent carbon dioxid and incubated at 37° C. for from four to five days. After forty-eight hours' incubation, abortuslike colonies were fished from the growth on the agar slants and replated. Final indentification was made by the agglutination and the agglutin absorption tests.

The remainder of the tonsil was ground in a sterile mortar with 5 cc. of sterile physiologic solution of sodium chlorid, which made a very heavy extract. A guinea pig was injected subcutaneously with 1 cc. of the extract. The guinea pig was killed and examined for evidence of Brucella abortus infection, from five to six weeks after injection. An agglutination test was made on the guinea pig's serum for abortus agglutinins. The tissues were examined for lesions of the disease and cultures made from the spleen and liver, as well as from other organs when they showed suggestive evidence of the infection.

Cultures were identified as Brucella abortus by the same methods as heretofore mentioned. The results of these examinations revealed Brucella abortus in either one or both tonsils from eight of fifty-six patients. In three patients, Brucella abortus was recovered from both tonsils, while in the other five it was obtained from only one.

This is hardly the place for discussing the therapy of brucellosis, as this disease is very rarely seen by otologists. I shall, therefore, only briefly mention the fact that cases treated in time with the serum melitensis have shown good results, especially if accompanied by intravenous injections of 5 cc. of a 40 per cent urotropin solution, with changes in the spinal fluid intralumbar injections of urotropin may be advisable. Blood transfusions and injections of normal serum have so far had little success. Some writers attribute some success to trypaflavin. Injections of convalescent serum may perhaps also be recommended. Figures published by Evans make it evident that brucellosis is a disease which is increasing. This is, furthermore, statistically confirmed by W. Lieberherr, who states that the percentage of staff cells in blood counts of the disease in the years 1927-1933 has increased every year at an average of from 3.2 to 23.6 per cent.

A. C. Evans writes in her article about chronic brucellosis that these cases show a picture of symptoms which are very similar to the textbook definition of neurasthenia: "Exhaustion, insomnia, irritability and aches and pains for which no objective signs can be found."

Evans, therefore, comes to the conclusion that "there is no doubt chronic brucellosis is often diagnosed as neurasthenia."

I would like to add that in acute, subacute and chronic brucellosis impaired hearing and impaired equilibrium with tinnitus and vertigo are apt to occur. Otologists should, therefore, in cases showing such symptoms, where other causes cannot be determined, consider brucellosis as a possible diagnosis. There is no doubt that even the otologist can frequently make the diagnosis.

SUMMARY.

Brucellosis is a disease occurring in man and increasing in frequency, and may appear with symptoms similar to acute infectious diseases, and also in the chronic form frequently similar to neurasthenia. Brucellosis is not diagnosed correctly as often as it should be, according to the frequency of its appearance. If physicians will be "brucellosis conscious," brucellosis will be diagnosed very readily, either by finding the brucella melitensis in the blood of the patient or, even simpler, by serum agglutination or by the abortion skin test.

In acute and chronic brucellosis, affections of the ear, pharynx and larynx and probably also the paranasal sinuses seem to occur. These complications have been observed until now, only in isolated cases. They will, in all probability, be more frequently observed if otologists remember to consider the possibility of brucellosis.

667 MADISON AVE.

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XXIV.

AN EXPERIMENTAL STUDY OF STIMULATION DEAFNESS IN GUINEA PIGS.*

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SEATTLE.

In a previous article,[1] an experiment on the effect of intense and prolonged acoustical stimulation on the auditory sensitivity of guinea pigs was reported. In that experiment seven guinea pigs were exposed for 110 hours to a tone of 1,000 cycles per second at an intensity of 125 decibels above the normal human threshold for that frequency. A small general loss of sensitivity was found for the eight octave-tones in the frequency range 64 to 8192 cycles per second, and this loss was no more confined to one tone than to another. It was also pointed out in that article that early investigators in the field of "stimulation deafness" reported that high frequency exposure tones produced degenerative changes within the cochlea more readily than did low frequency tones. Von Eicken,[2] in his review of the field, stated that if a tone was not high and shrill, the length of the exposure period was a factor in the amount of degeneration produced.

In the present experiment two new groups of guinea pigs were studied. One group (eight animals) was subjected to a tone of 1,500 cycles per second at approximately 100 decibels above normal human threshold for 2,000 hours, and the other group (four animals) was exposed to a tone of 3,000 cycles per second at approximately 100 decibels above normal human threshold for 1,250 hours. The auditory sensitivity of each animal was studied before and after the exposure period by employing the conditioned respiratory response method as outlined in a previous study.[3]

APPARATUS AND PROCEDURE.

The apparatus consisted of a Western Electric 2A audiometer, an amplifier, an 8-volt D. C. loud speaker located within a sound- and light-proof structure in which the animal was placed during tests, a cradle-like holder for the animal, photographic devices for recording

*This investigation was carried out at the Psychological Laboratory of Princeton University.

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the animal's breathing, and equipment for shocking the animal. Each animal was trained to respond regularly by a change in breathing on the appearance of a loud tone. A sensitivity curve for each animal was then obtained in the following manner. An animal was presented with a loud tone whose intensity was lowered at successive intervals in steps of five decibels to a point well below audibility. The intensity was then raised in the same manner. The lowest positive response was noted for future comparison. This test was made on each of the eight octave-tones in the frequency range 64 to 8192 cycles per second. In addition to these tones, measurements were made on the tone of 1,500 cycles per second for one group, and on that of 3,000 cycles per second for the other group.*

After these measurements were obtained, each group of animals was exposed to its particular stimulating frequency for ten hours daily. The exposure period of the 1,500-cycle group lasted 200 days (November to June), and that of the 3,000-cycle group 125 days (June to October). Each exposure tone was produced by an audio-oscillator, and led through an amplifier to the loud speaker. The B-current of the oscillator and the amplifier was produced by an A. C. rectifier. This may have produced a slight A. C. "hum" in tone, but it was not audible when the tone was turned on. The intensity of the 1,500 cycle tone, as measured by a condenser-microphone, was 56 dynes/cm,^{2} and that of the 3,000 cycle tone was 36 dynes/cm.^{2} Thus the intensity of both tones was about 100 decibels above normal human threshold for each frequency. Such intensity is above the threshold of feeling.

The guinea pigs were placed inside the sound- and light-proof structure during the daily exposure period. This box was so constructed that, during the testing period, the walls were lined with thick layers of absorbent cotton to minimize standing waves. For sanitary reasons it was impossible to retain the absorbent cotton lining during the exposure period, and therefore a second method of controlling standing waves was adopted. This consisted of a three-bladed fan, revolving at the rate of once every three seconds, which continually changed the conditions of sound reflection within the enclosure. Examination of the structure by sound-measuring instruments revealed no "dead spots". Thorough shielding of all leads avoided inductive effects.

At the beginning of the first few daily exposure periods, both groups of animals appeared frightened, but within fifteen to twenty

*For a more detailed description of this apparatus and procedure, see Horton.[3]

minutes they were moving about, eating and drinking in a normal fashion. This initial stage of fear disappeared within a few days, and during the rest of the exposure period there was no observable response to the loud tone. This complete adaptation to the intense sound was not present in the author's previous experiment, but was reported by Wittmaack[11] in his investigation. The greater intensity used in the previous experiment may account for the difference in the behavior of the experimental groups.

At the end of the exposure periods the animals were retrained, and when they again responded regularly to the various frequencies they were given the same tests that they had received previous to the exposure period. The results of these tests appear in figures 1 (1,500-cycle group) and 2 (3,000-cycle group). The lowest positive response that an animal exhibited to a given tone after the exposure period is compared with the lowest positive response that it exhibited to the same tone previous to the exposure period. The responses occurring before the exposure period are indicated by the zero line. The various frequencies are plotted along the abscissa, while the ordinates represent units of loudness (decibels) above and below the loudness level to which the animal responded positively before the exposure period. The units above the zero or normal line indicate increased sensitivity, and those below decreased sensitivity. It will be noted that in some cases the graphs are not continuous. There were tests on which some animals did not exhibit positive responses, or on which the record was so poor that the experimenter did not consider it reliable. When, as sometimes happened, an animal was not securely fastened in the holder, the breathing record proved extremely difficult to read. If for any of these reasons a record was not considered reliable, it was left out of account.

Three animals of the 1,500-cycle group died during the exposure period. Their cochleas were fixed and delivered for histologic examination. Animals 56 and 86, whose graphs appear in Fig. 1, were born during the exposure period. They were subjected to stimulation along with the others, and the length of the exposure (after birth) was 1,070 hours for No. 56, and 1,000 hours for No. 86. The sensitivity of these two animals after the exposure period is compared to the average original sensitivity of nineteen other animals used in this and the previous experiment.

The results of the tests made at the end of the exposure period are indicated in both figures by the solid line. As in the previous experiment, there is a general loss of sensitivity to all tones, and the loss is no more confined to one tone than to another. In some cases

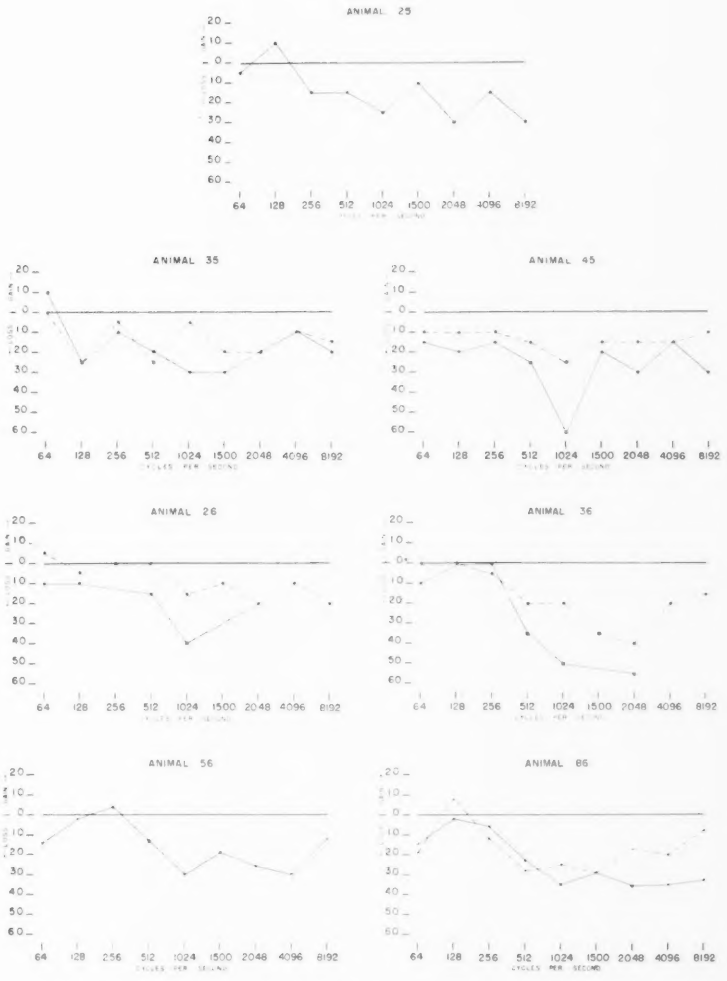

Fig. 1. 1500-cycle group: Sensitivity after exposure-period (solid line) is compared with sensitivity before exposure began (zero line). Broken line indicates sensitivity three and one-half months after the end of exposure-period. All animals had 2000 hours stimulation except 56 and 86, which had 1070 and 1000 hours respectively. Numbers 26 and 36 are females, the rest males.

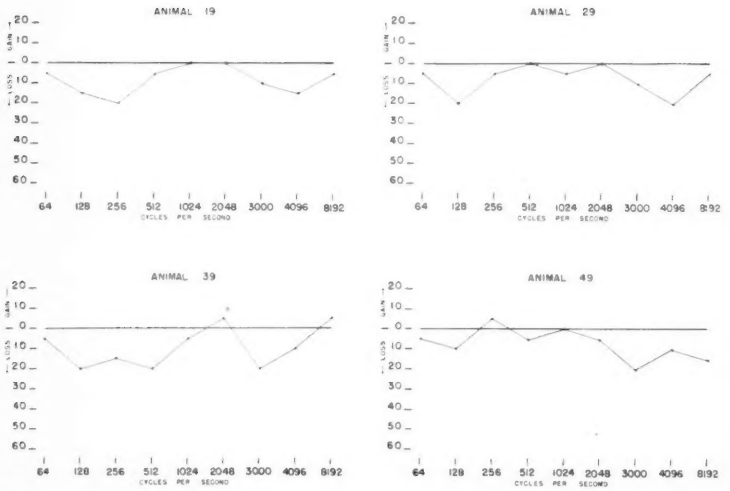

Fig. 2. 3000-cycle group: Sensitivity after exposure period (solid line) is compared with sensitivity before exposure began (zero line). Exposure-period was 1250 hours. All females.

animals responded to the original intensity level, and in others they responded to even weaker intensity levels. In these latter instances the original responses were high in comparison with the other animals, and it is probable that a more thorough investigation of these thresholds would have shown them to be lower than indicated.

About three and a half months after the exposure period the 1,500-cycle group was retrained, and on the 127th day after the exposure period a second set of tests was begun. Animals 25 and 56 were sick at the time, and could not be given the tests. The results appear in Fig. 1, and are indicated by the broken line. The same general loss of sensitivity is again found, and, as before, it varies from animal to animal and from tone to tone. The results agree with those found in the previous study. In that experiment the possibility of recovery was studied over a period of ten months and no definite recovery was noted.

COMMENT.

In the three groups of guinea pigs the results have been the same, namely, a small general loss of sensitivity which varies from animal to animal and from tone to tone. This result may be produced by an extremely intense tone, e. g., 1,000 cycles per second at 125 decibels, acting for a relatively short time, or it may be produced by an intense

tone, e. g., 1,500 cycles per second at 100 decibels, acting for a long time.

The results of the author's experiments are not directly comparable to the early work in the field of "stimulation deafness", since those of the previous investigators are in terms of degenerative changes and not changes of sensitivity. Nevertheless, some of the previous experimenters inferred serious losses of sensitivity from the histologic changes which they found. The small general loss of sensitivity is not parallel to the results of the former workers who reported that high tones produce greater degenerative changes in the cochlea than do low tones. The high tone of 3,000 cycles per second did not produce changes in sensitivity which are significantly different from those produced by the tone of 1,500 cycles per second at the same intensity level. The results of the 1,500-cycle group do not bear out von Eicken's statement that if a tone is not high and shrill, the length of the exposure period is a factor in the amount of degeneration produced. The two animals of this group, which were exposed for only 1,000 and 1,070 hours respectively, show the same general loss of sensitivity as the five animals which were exposed for 2,000 hours.

Some of the previous experimenters tested the auditory ability of their animals before and after prolonged exposure to sound. Marx[6] used Preyer's reflex as a sign of hearing, and Upton[11] and Finch and Culler[2] employed conditioned response technics. Marx reported that Preyer's reflex in guinea pigs disappeared after prolonged acoustical stimulation. In the present experiment Preyer's reflex, as elicited by a Galton whistle, was observed both before and after the daily exposure period. In the 3,000-cycle group it was always present, and in the 1,500-cycle group it was present with occasional exceptions during the entire exposure period. In the author's previous work it was absent during the entire exposure period, but was again present one month after the exposure period. These observations, coupled with the data furnished by the conditioned respiratory response method, show that the mere presence or absence of Preyer's reflex is no true indication of a guinea pig's auditory acuity. Moreover, they confirm the opinions of von Stein[8] and Röhr[7] as to the unreliability of this reflex as a sign of hearing.

Upton reported a great loss of sensitivity to the exposure frequency. He was unable to recondition two guinea pigs to the same intensity level of the exposure tone to which they had responded positively before the stimulation period. He does not report an attempt to train them to a greater intensity level. The results of the author's previous study did not confirm Upton's findings, and

it was suggested that the disagreement of results might be attributed to the fact that Upton had exposed his animals for 1,655 hours while the writer's 1,000-cycle group had only been exposed for 110 hours. However, in the present experiment the 2,000-hour period exceeds Upton's exposure period, and, moreover, the intensity of the exposure tone (100 decibels) is certainly greater than that used by Upton.

Finch and Culler, employing a conditioned leg withdrawal method, report a "horizontal subsidence" in auditory acuity in two dogs. One dog, exposed to a continuous tone of 1,000 cycles per second at an intensity of 110 decibels above the animal's limen, showed a loss of from 40 to 50 decibels throughout nine tones in the frequency range 200 to 5,000 cycles per second. When the stimulating tone was interrupted 52 times a minute, and the animal exposed for an additional 148 hours, the acuity dropped an additional 30 decibels. A second dog, exposed to an interrupted tone of 3,000 cycles per second at an intensity of 110 decibels above its limen, showed a loss of 56 to 79 decibels after 50 hours' exposure. The dog showed no recovery thirty-one days after the end of the exposure. The results obtained by the present author agree in general with those of Finch and Culler, but no such great losses in acuity as those of Finch and Culler have been found. These investigators did not retrain their animals before testing them, but to avoid extinction of the conditioned response they placed the second dog under complete general anesthesia during the daily ten-hour exposure period. The difference in the amount of loss may be due to the difference in the animals employed.

The results of this experiment and those of the previous one appear to support a nonresonance or telephone type of auditory theory rather than a simple resonance or place type. The small general loss of sensitivity rather than a specific loss to the exposure frequency suggests that some general change has taken place in the ear. This may be along the basilar membrane, or a change in the middle ear. For example, a loss of tonus of the muscles controlling the ossicles might cause a general loss of sensitivity. Hughson and Crowe,[5] studying the physiology of the cat's ear by the method developed by Wever and Bray, reported that sufficient tension exerted on the tensor tympani will completely block the transmission of sound up to 2,048 cycles per second. Tension may be so regulated that transmission will occur at a reduced level.

It is planned to study the auditory mechanism of these animals by the auditory nerve method used by Wever, Bray and Horton.[10]

An histologic examination of the mechanism will then be made. Any definite conclusions regarding the reasons for the change of sensitivity obtained in these experiments must await these studies.

SUMMARY.

Employing the conditioned respiratory response method, the auditory sensitivity of two groups of guinea pigs has been studied before and after prolonged acoustical stimulation. A small general loss of sensitivity which varies from animal to animal and from tone to tone has been found.

Tests made three and a half months after the exposure period reveal no recovery from this loss.

The author wishes to express his appreciation of the assistance given by Professor C. W. Bray, Doctor C. H. Wedell, and Mr. W. Habada in conducting the daily exposure period in his absence. He also wishes to thank Professors E. G. Wever and C. W. Bray for measuring the intensities of the stimulating frequencies, and for other assistance with the apparatus. The condensor microphone was kindly loaned by the Bell Telephone Laboratories.

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XXV.

A NEW SOUND COUNT FOR LOGOPEDISTS.*

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The laliatrist is interested in the frequency of occurrence of phonetic sounds as an index to the etiology of certain speech pathologies. That is, if a sound is seldom heard, that fact in itself might be sufficient to influence the patient to utter that sound in terms of his more common experiences and in that way develop a speech defect of the type often referred to as phonetic substitution, a sort of dyslalia. The speech corrector uses this information, the sound count, in developing corrective exercises and the teacher of speech as a basis for the building of the speech curriculum.

Dr. Lee Edward Travis, director of the Psychological and Speech Clinic at the State University of Iowa, made a study of the frequency of occurrence of speech sounds, in 1931, because, as he says, "Possibly a child has difficulty with a sound largely because he seldom hears it."[1] However, the Travis count, based on 16,077 sounds, includes only the consonants.

Dr. E. B. Twitmyer, director of the Psychologic and Speech Clinic at the University of Pennsylvania, probably had this sort of dyslalia in mind, among other things, when he had his assistant, Dr. Nathanson, develop a series of therapeutic exercises on the basis of Dewey's sound count,[2] which used dictionary pronunciation for 385,912 sounds from passages selected from written or printed material. No phonetician would deny that any sound count should be based on spoken language, and hence the logopedist's therapeutic exercises should be based on actual speech.

This paper attempts to present a sound count based on speech usage. The type selected in this study, for American pronunciation, was the speech of radio announcements, broadcast from radio stations distributed over the entire United States. From these radio stations in all sections of the country, 5,946 announcements were recorded phonographically, directly from the radio, and studied.

Not only are the radio stations themselves widely distributed, but their announcers, as they happened to be in the case of the selec-

*Speech and Hearing Clinic, Ohio State University.

tion in this study, appeared to have had their speech influenced by almost all the American dialects, including such fine divisions as "Pennsylvania Dutch" and "French Canadian English." Also the wider English speech dialects as "speech of Hawaii" and other islands of the Pacific, "London cockney" and the speech of a number of sections of England, Ireland, Scotland as well as Canada. Many of the announcers had been born in foreign countries, learned to speak another language before English, or were educated in other languages or studied abroad; or perhaps their parents were born in foreign countries or spoke other than the English language in their home; and of course many of the announcers speak more than one language. Dialectal English as well as foreign tongues have had their influence in the speech of these people.

All of them were trained speakers, and many had had a great deal of speech education. To illustrate some of the training listed by the announcers in reply to my personal letter or my questionnaire, they listed the following types of speech training: Practice as a teacher of speech, theatrical experience, training in diction, phonetics, dramatics, public speaking, English, voice and singing. The fact that many were children of lawyers, ministers or teachers cannot be underestimated in the influence this might have had on their speech. One even mentioned that correct speech had been a game of his childhood with just rewards for his success.

I wish I had space to comment on the personalities of the announcers which would give a whole list of speech influences. However, having read their letters and heard records of their voices, I can easily say that a multitude of different types of personalities are represented in my selection.

From the point of view of speech, it is of interest to find that the selection included people from almost every level, from ministers' sons, which Terman lists as the highest, on down to many much lower classes. But many were college trained.

Some announcers had lived rural lives, others urban; some still lived in their original environment but many had lived in a great many different places. The residences of the announcers were an important index of geographical speech influence, and further proof of the comprehensiveness of this selection of American speech. Incidentally many had traveled considerably, especially with theatrical troupes.

Perhaps the greatest advantage for using the speech of broadcasts for my purpose was that the announcer had no opportunity to change

THE FREQUENCY OF OCCURRENCE OF SPEECH SOUNDS AS A BASIS FOR SOME TYPES OF DYSLALIA.

Phonetic Sounds	Key Words	Relative Frequency Per Cent
I	pip	7.88
n	nip	7.32
t	tip	7.20
ə	above	6.80
r	rip	6.49
d	dip	5.11
s	sip	4.66
ɑ	pop	4.15
l	lip	3.90
ð	the	3.16
æ	pap	3.09
ɛ	pep	3.05
ɪ	peep	2.83
m	mit	2.76
k	kick	2.56
f	fit	2.15
z	zip	2.14
ɔ	pawp	2.13
o	pope	2.12
e	pape	1.99
u	poop	1.99
b	bit	1.96
w	wit	1.84
ʊ	put	1.76
h	hit	1.64
v	vim	1.54
p	pip	1.48
j	yes	1.16
g	go	1.08
ŋ	ping	1.03
ʃ	ship	1.01
θ	thin	.65
ʌ	pup	.42
ʒ	azure	.41
ʍ	whip	.37

his pronunciation in the study, because he was unconscious of our recording of his speech until afterwards. Besides, he was not removed from an environment natural to him.

Hence, the speech of radio announcers might be considered as approaching the normal for typical formal American pronunciation. They tend to follow and to some degree might conceivably have some influence in guiding speech trends.

To secure a diversity of material, station announcements were not used. The selected programs were only from advertising, informative or entertaining programs.

The programs broadcast were recorded phonographically and studied from the record, being transcribed into the international phonetic alphabet by agreement of several independent workers.

Since the announcements were extremely brief, a selective continuum of 100 sounds was taken as a minimum length. In all passages, 660,594 sounds appeared.

The accompanying table shows the sounds tabulated and listed from the most to the least frequent in occurrence, giving their relative frequency per cent.

The logopedist can, at a glance, perceive which sounds are the more susceptible to phonetic substitution, as a result of the very uncommonness of these sounds; and which sounds are the most likely to be used in their place.

1452 North High Street.

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XXVI.

THE MANAGEMENT OF RECENT FRACTURES OF THE NOSE AND SINUSES.*

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The management of recent fractures of the nose and sinuses has become a real problem, and is becoming more so as modern high-speed transportation is developed.

Until recently the occurrence of this type of injury was more or less due to blows of direct violence encountered in the ordinary pursuits of life. When these injuries did occur, they were of a more minor nature than those seen today.

The automobile is largely responsible for the great increase at the present time, which can be readily proven by examination of the automobile accident statistics of recent years, and the personal experience of all who have these injuries to treat. It is also true that when this type of accident does occur, the resulting injury is far from a minor one, for in most instances these fractures involve not only the nose proper, but one or more of the accessory sinuses, and quite often the skull, brain, orbit, eyes and jaws.

For the above reasons the responsibility has fallen upon the oto-laryngologist, instead of the general surgeon, and properly so, for no one is better equipped to handle them than one whose daily work has given him an intimate knowledge of the anatomy and treatment of conditions in this area.

While the treatment is primarily his responsibility, he must work in association with the neuro-surgeon, the ophthalmologist, the orthodontist and the dentist, for their combined special knowledge and skill are often called upon during the treatment of these severe fractures.

When one confronts these problems in his daily practice, he is handicapped by the lack of unanimity of opinion as to the procedure

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*University of Virginia.

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to be followed, for we have not yet compiled sufficient data from the articles published, in widely scattered journals, to serve as a guide.

Initial treatment is largely concerned with control of hemorrhage, either by local packing, ligation of bleeding vessels, or ligation of the efferent artery at some point distal to the wound.

The treatment of shock is very important, since it is so often present in marked degree in the more severe cases. All attempts at repair should be postponed for the time being, for lack of consideration of this condition may well bring about the death of the patient.

One should also consider the type and condition of the wound, for this is the time to administer tetanus and gas bacillus antitoxin, with due consideration, of course, for the patient's sensitization to horse serum, which in many instances creates an additional problem at this time.

The examination should be carried out in great detail in regard to repair and the patient's general condition, for if we are suspicious of intracranial injury it is quite important to keep a record of the blood pressure, respiration and pulse as well as to note if there is a discharge of spinal fluid from the wound. It might well be that treatment of intracranial injury is of primary importance, and neurosurgical consultation should be sought, or again, there might well be some serious general surgical condition which should receive primary attention.

Granted that these possibilities do not present themselves, then we may continue with our own consideration of the problem. First, inspection will tell us whether deformity is present and with palpation, should give us a rather clear picture of the type and degree of injury, as well as an outline of the procedure to be followed in its repair.

Transillumination of the sinuses may be sufficient in the less severe cases; otherwise, roentgen ray studies of the skull and sinuses should be done. While it is true that the multiplicity of lines and shadows in this region makes interpretation difficult, still careful consideration and measurements often show clearly the type and degree of fracture present, and presence of blood or infection in the various sinuses. Shea[1] has shown the value of the roentgen ray in fractures of the malar bone and zygomatic process.

Complete repair can usually be done at this time, but in some cases we must be content with temporary repair after due consideration of future plastic procedures. When this is true, then it is important to conserve as much skin, mucous membrane and bone as possible, in relation to the future steps in plastic surgery. For instance,

the suturing of skin to mucous membrane, the anchoring or transposition of viable bone fragments, and the maintenance of adequate blood supply are all of importance at this stage.

Local anesthesia is usually sufficient, using cocain or some of the newer synthetic preparations as a topical application, and procain subcutaneously. At other times it may be best to use gas, ether or avertin, depending on the particular type of case and the surgeon's judgment.

The danger of vomiting after wiring the teeth together should always be considered, and every precaution should be taken to prevent it.

Very few special instruments are necessary in addition to the ones commonly used in otolaryngology. I would suggest Ritter sounds, a pair of the old Adams or Asch forceps, Gill's heavy towel clip for malar bone fractures; also a stout hook and a Robert's corkscrew for the same purpose, one or two heavy elevators and a drill for wiring bone fragments.

When infection has set in, adequate drainage along the usual lines must be instituted, and cultures should be made, for we are particularly afraid of the staphylococcus, since it is the usual cause of osteomyelitis.

As a result, however, of recent work on the staphylococcus, we are less fearful than we used to be, for the patient's blood titer against the staphylococcus may be estimated, and his defense may be enhanced by staphylococcus toxoid in certain cases and the use of staphylococcus antitoxic serum[2] in others.

The importance of repeated blood transfusions in severe infections and injuries is widely employed and merely needs to be mentioned at this point.

After due consideration of all preliminary studies, the time of repair must be considered in regard to swelling and the patient's general condition. As a rule, these fractures can be readily reduced up to a week or ten days, yet the earlier reduction is done the better.

The details of treatment will be discussed under each particular structure. Generally we might say that it largely resolves itself in the reversal of the direction of force with adequate fixation of the fractured parts and at the same time suture, debridement and restoration of the soft parts to restore function and prevent deformity.

It should also be remembered that practically all of these fractures are compound, in that they communicate with the outside

world either externally or internally through the nose and sinuses or both, so that due regard to subsequent or existing infection must be considered in each case, as well as for interstitial emphysema and hematomata.

THE EXTERNAL NOSE.

The rigid elements are composed of the nasal bones and the nasal processes of the maxillæ, except in a few cases of congenital variation. The remainder of the nose is composed of resilient cartilage, and except for dislocation and displacement at its bony junction it very seldom enters into the deformity, so the problem largely resolves itself into reduction of the displaced bony elements by reversal of the fracturing force. Since the deformity is evident, firm pressure with the surgeon's thumbs will readily correct the dislocation, and the result immediately ascertained; in some instances it may be necessary to employ an elevator intranasally, or the Adams forceps, especially in those cases where there is a depression of the bony parts.

Reduction is usually accompanied by a sharp click as fragments assume their normal position, and seldom is splinting necessary. We merely depend upon a piece of adhesive across the nose, with or without a small roll of gauze underneath, to remind the patient and his friends that the nose should not be touched during the healing period.

In the more severe cases of compound fracture, there may be an actual loss of bone and soft tissue. It is in these cases that immediate repair should consider future plastic work, such as the introduction of rib cartilage, tube grafts, the conservation of skin and mucous membrane, and any viable bone fragments; therefore, each case is a law unto itself, and only general principles need be considered.

Debridement should be carefully done in all lacerated wounds, and the proper placing of subcutaneous catgut and skin sutures will materially lessen deformity and scar.

THE NASAL SEPTUM.

The septum is practically always involved in all fractures of the external nose, and in many instances, when there is no evident fracture of the external nose, even infants and young children often present deformities of the septum which can only be explained by blows on the nose, which were disregarded at the time, and then forgotten. These injuries undoubtedly account for many of the septal deformities seen in later life.

When one considers the component parts of the septum, namely, the perpendicular plate of the ethmoid, the vomer, the septal carti-

lage and the vomeronasal cartilages, he readily understands why blows on the nose produce deformity at the junction of the vomer with the other septal parts. Unfortunately, even when this condition is recognized at the time of the injury, very little can be done to correct it, because of the resilience of the cartilages. They are nearly always very difficult to reduce or to hold in place after reduction, so in spite of our best efforts we must often depend on future submucous resection to restore function.

Hematoma is often found after these injuries, and when large enough to produce obstruction should be drained by incision through the mucous membrane, for otherwise they tend to become infected, or to heal with a resultant fibrous mass of scar tissue which produces obstruction to aeration.

THE FRONTAL SINUS.

Fractures involving the frontal sinus are potentially the most dangerous of all, because of the anatomic relationship to the brain and meninges. It is not uncommon to find these fractures extending into the vault or base; hence neurologic vigilance is important. Fractures into the orbit are also frequently seen, which menace the important structures contained therein.

As complications, we may look for meningitis, brain abscess, orbital abscess and osteomyelitis as the most serious, and it is primarily to prevent these that early and adequate treatment is of such importance.

It should always be borne in mind that fracture here may involve the anterior wall, the posterior wall or both. Therefore doubtful cases should be explored surgically to determine the exact extent of injury, to remove bone fragments and to institute adequate drainage.

When the wound is open externally we are usually able to inspect the sinus through the original wound. Otherwise we advise the usual curved incision through the unshaven brow, and inspection by removal of the floor of the sinus, which leaves very little scar and no deformity. It is only by adherence to this apparently drastic principle that we can prevent future serious complications.

When it is found that the fracture involves the anterior wall only, and presents no deformity and no injury to the integument, no treatment may be necessary, other than close observation during the healing stage.

Blood clot may be present within the sinus in many cases, but it may also be readily absorbed, and need not be considered a cause

for worry. However, if at any time during the healing phase we should become doubtful as to the condition of the sinus we should advise exploration.

When a frontal sinus is explored either externally through the original wound or by operation through the brow, we should carefully remove all blood clots and loose fragments of bone, then inspect the posterior wall for possible injury, and see that adequate drainage is established, either externally if the original wound is suitable, or preferably intranasally by a rubber tube inserted through the nose to the depths of the sinus, and allowed to remain in situ for from ten to fourteen days.

In the presence of large anterior defects, external drainage is justifiable, conserving all bone and periosteum possible to prevent deformity. In all other instances we advocate intranasal drainage.

When the posterior wall is found to be involved, the entire mucous membrane should be carefully removed, and the edges of the fracture carefully inspected. In these cases our procedure is analogous to similar situations encountered in mastoid surgery—that is, to remove bone fragments from the dura and to enlarge the defect until we are sure we have healthy or rather uninjured dura. Should the dura be torn, external drainage is imperative, and communication with the nose is blocked off by packing in the region of the ostium. After Nature has walled off this area, we may do a secondary closure of the external wound and concern ourselves with the cosmetic result.

Suturing dural tears, and in some cases the interposition of fascia lata has been advocated, but in our experience neither of these has been necessary. Fortunately the dura is a wonderfully resistant membrane and many lives are saved by this wise provision of Nature.

When the posterior wall is fractured but the dura is uninjured, our procedure is the same, except that we employ intranasal drainage.

All patients with fractures involving the accessory sinuses should be cautioned about blowing the nose, to prevent interstitial emphysema, but this warning is particularly important when the frontal and ethmoid sinuses are injured.

As has been previously stated, neurologic vigilance is important. It might be added that bacteriologic vigilance is also important, not only to prevent infection but to determine the type should it occur.

In our experience with brain abscess and osteomyelitis, the staphylococcus is nearly always the offending organism. We are constantly on watch for it, in order to protect our patient as early

and as well as possible, first by estimating his blood titer of resistance against the staphylococcus and then employing either toxoid in certain instances or antitoxin in others. In our limited experience it seems that there is a great future to this line of treatment in staphylococcus infections. For futher information I would suggest reading the papers by Dolman of Toronto, which reference is given in the bibliography.

THE ETHMOID SINUS.

The ethmoid is involved more often than any other sinus, and while the potential danger of complications is great, fortunately we do not meet them as often as in frontal sinus fractures.

The relationship to the brain and meninges and the orbit must be remembered, and it is of particular importance here to caution the patient against blowing the nose, because of the likelihood of creating an interstitial emphysema, and thus increasing the danger of meningitis and orbital infection.

Fractures through this region seldom present much deformity, so correction of the external nose deformity in the milder cases is usually sufficient. In those of severe injury with loss of tissue, we can restore some order by working through the external wound; and in those of transverse facial fracture, through the ethmoid and orbit, apposition is obtained by anchoring the whole fragment either by wiring or with dental braces anchored to a plaster skull cap.

Nature has wisely furnished an abundant blood supply to all of the nose and face, and with the high degree of tissue immunity to infection also present, many discouraging cases often heal with surprisingly good results.

It may be necessary to remove sequestra or to drain a few ethmoid cells at some later date, either externally or intranasally. However, it is usually a comparatively simple procedure and a good final result is obtained

THE SPHENOID SINUS.

Due to its protected position, fracture of the sphenoid bone is fortunately rare, except in the most severe injuries. Then it is more likely that the greater and lesser wings, or the pterygoid processes, in some transverse facial fractures, are involved; and the other problems presented in severe injury overshadow the problem of the sphenoid.

Penetrating wounds such as those caused by sharp objects and bullets do occasionally involve the sinus itself, and it may be neces-

sary to search for foreign bodies or to see that proper drainage is instituted.

One must bear in mind the intimate relationship of the sphenoid and posterior ethmoid sinuses to the optic nerve, for we have occasionally seen optic nerve atrophy follow injury to the head, which could only be accounted for by fracture in this region which was not recognized at the time.

MAXILLARY AND MALAR BONE.

These two bones are so intimately associated that fracture of one very often extends into the other, and in either case the maxillary sinus and orbit are usually involved.

The malar bone forms the most prominent part of the face and is therefore most frequently injured, and since it articulates with the maxilla, the frontal, the zygomatic process of the temporal and the sphenoid, its attachment is essentially weak and dislocation easily takes place. By studying its location and shape in the dry skull we can visualize the result of blows from various angles. This point is well illustrated by Gill[3] in his excellent article on this subject.

The maxilla largely houses the maxillary sinus, and also forms a prominent part of the face, so is likewise subject to frequent fracture. This may take place from extraction of teeth, from direct blows of a crushing nature over the body, from blows on the front of the face producing transverse facial fractures, either through the alveolus, the nose and bodies of the maxillæ or through the orbits. Blows on the nose often fracture the nasal processes of the maxillæ and blows on the malar secondarily involve it. So in this brief résume we can readily see that all of us should review our anatomy and study the many excellent articles already published on the correction of these fractures.

Diagnosis largely rests on inspection and palpation, aided by our knowledge of anatomy and mechanics. Roentgen ray studies are very valuable here, and no attempt at reduction should be made until the films have been carefully studied and a mental picture of the deformity firmly impressed on our minds.

In nearly all cases we find the sinus contains blood clot. In simple cases with no deformity it will frequently be absorbed in a few weeks, and it is unnecessary to wash the clot out, unless infection takes place.

In the more severe cases we are in no hurry to irrigate the sinus, preferring to wait until Nature has walled off all fracture lines before doing so. Usually four to six days are sufficient; then we run no risk

of introducing and spreading infection. In other cases that are infected in which we wish to use an intranasal route to reduce dislocations, we are content with a large drainage opening in the inferior meatus, for the time being, and only employ irrigation after the four to six day interval.

Treatment, of course, depends on the type of injury. Generally it is a question of reversal of the fracturing force by whatever method happens to be best suited to the particular case and the prevention of deformity.

The orbital rim must be restored as well as the contour of the orbit, so in certain cases it may be necessary to make an incision along the rim to restore contour, to remove blood clots from the orbit or to drain orbital abscess. In comminuted fractures of the anterior wall where there is an external wound into the sinus, we may be able to work through this defect, removing clots and fragments, and utilizing all viable fragments in restoring contour, after which the external wound is carefully repaired and intranasal drainage instituted.

The reduction of dislocated malar bones and zygomatic processes is usually readily accomplished in several ways: The towel clip method of Gill, the hook under the process, the intra-antral method of Shea or by the method of Gillies. All of these methods and others are well discussed in numerous papers on this subject, so it only becomes necessary for us to familiarize ourselves with these various methods and to include the necessary instruments in our armamentarium, so that they may be available when the necessity arises.

The correction of transverse facial fractures is usually best accomplished by wiring the teeth and the application of various orthodontic appliances anchored to a plaster skull cap. Ivy[4] well illustrates these in his book on this subject. For those of you who unfortunately do not have consultants of this character to call upon it would be well to study these methods in great detail.

In high transverse facial fractures through the orbits, Gill wisely advocates wiring of the zygomatic process of the frontal to the frontal process of the malar by open operation, which should be a logical and useful procedure in many cases.

Wiring of fragments is necessary in many instances of fractures about the face, as it gives firm anchorage and materially shortens convalescence.

Fractures through the alveolar border and hard palate, involving the teeth, frequently accompany these severe injuries, and the ortho-

dontist and dentist should be called upon in all these cases to correct deformity and to restore function.

CONCLUSION.

In conclusion, I merely wish to reiterate what has already been said: That although the responsibility for the treatment of these fractures falls primarily upon the otolaryngologist, the problems are so numerous and involve so many highly specialized fields that he should work in close co-operation with the surgeon, neuro-surgeon, ophthalmologist, faciomaxillary surgeon, orthopedist, orthodontist, dentist and quite frequently the pediatrician and internist, which constitute quite an array of specialized knowledge, but illustrates very well the complexity of this problem.

104 EAST MARKET ST.

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XXVII.

THE PRESENT STATUS OF SINUS INFECTION AS AN ETIOLOGIC FACTOR IN RETROBULBAR NEURITIS.*

JAMES A. FISHER, M. D.,

ASBURY PARK, N. J.

This old subject, concerning which volumes have been written in recent years, is still without any conclusive evidence as to the procedure to follow when confronted with such a problem.

It is with fear and trepidation that I approach the subject, but several articles having recently appeared in medical literature which gave me grave thought that the pendulum might swing too far from the sinuses as a causative factor in this condition. i. e., retrobulbar neuritis.

Quoting from the late Ross Hall Skillern, "The intimate relation of the sphenoid sinus with the optic nerve is a predisposing factor to retrobulbar neuritis following sphenoid sinusitis, particularly if a dehiscence is present. No regularity exists as to the thickness of bone separating the sinus from the nerve. Investigations have shown that these measurements undergo great variations in different heads and even on different sides of the same head, as in one instance the nerve may be almost in direct apposition to the mucosa of the sinus, while on the opposite side several mm. of spongy bone may intervene."

The posterior ethmoid or spheno-ethmoid cells are also in very close proximity to the nerve.

A number of observers have examined a series of patients with spheno-ethmoiditis for signs of involvement of the optic nerve without any complaint on the part of the patient of ocular symptoms. Markbreiter found that 70 per cent of a series showed defects in the fields of vision, and Bordley found enlarged blind spots in 31 of 102 patients examined.

With this evidence before us, it is reasonable to assume that we are not dealing with a possibility as remote as some authors would have us believe.

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*Presented before the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Portland, Me., January 4, 1935.

Von Hippel[1] asks the question, "Are the successes of nasal therapy actually the result of nasal operations or are they produced by spontaneous recovery?" He believes the latter to be the case. He bases his conclusions on a series of thirty-six cases; in thirty there was no pathologic condition in the sinuses by roentgen ray examination. The remaining six cases were definitely either multiple sclerosis or hereditary optic atrophy. Twenty-one cases recovered; the remainder pursued an unfavorable course or failed to report. None were operated upon; thus surgery was prevented from scoring a 70 per cent success.

Benedict[2] states: "When one considers the vast number of cases of severe suppurative sinus disease without visual symptoms the connection between sinus disease and retrobulbar neuritis becomes much less credible."

He gives in support of his opinion an analysis of the etiology of 225 cases of retrobulbar neuritis at the Mayo Clinic, in which series but one case was considered as due to sinus disease. He believes that the therapeutic value of operation on the sinuses does not lie so much in the eradication of the infected area as to the immediate ischemia from blood letting followed by a secondary congestion of the adjacent tissues until healing is complete. "If the operation has been sufficiently extensive, there is commonly a rise of temperature of one to two degrees from the absorption of blood which, in effect, is autovaccination. These two factors, congestion and absorption, are responsible for any improvement noted."

Blatt[4] found the number of cures obtained in patients on which operation was performed was no greater than the number obtained in patients on which no operation was performed. Nor was the course of the disease influenced by operation. He advises surgical intervention in apparently negative cases where no results have been obtained by other means after a period of two to three weeks.

However, let us contrast a report by Bajkay,[3] who reviewed forty cases in which the ethmoid and sphenoid sinus were opened, where rhinologic examination gave positive results in twenty-two instances and negative in eighteen. In both of these groups operation produced a complete cure in those patients who entered the clinic in from three to four days after the onset of visual disturbances, but in those who entered between the eighth and fourteenth day after onset of symptoms only a partial restoration of vision could be obtained. In eight cases there had been a delay of from three to four weeks before admission and no improvement resulted.

Bachstez[5] states that while rhinogenous retrobulbar neuritis is not rare, rhinogenous cases with negative rhinologic findings are rare.

There have been cases immediately following epidemics of influenza in which multiple sclerosis could be definitely ruled out. Feigenbaum and Salzburger[6] reported a series of twenty-four cases, twelve of which showed x-ray evidence of sphenoid and ethmoid sinus infection. Sixteen were operated upon; of these, eleven were cured. The fact that the major portion of these cases occurred about four weeks following influenza seems to be reason for considering that the etiology was nasal in this particular group.

In order that I might have the most recent opinion of leaders in ophthalmology and rhinology in this country, I addressed an inquiry to men in various sections. As might be expected, those agreeing that sinusitis was an important factor in retrobulbar neuritis were, for the most part, rhinologists and to a lesser number some ophthalmologists. Each man was asked for his present attitude in regard to the etiologic factor of spheno-ethmoid infection in retrobulbar neuritis.

O'Brien of Iowa City said: "I have seen very few cases of retrobulbar neuritis which I feel were due to sinus disease. I am not satisfied that an operation on the sinuses is indicated in those cases which have slight evidence of disease."

Edward Jackson of Denver answered: "In cases of monocular neuritis I have not advised opening of the sinuses; and in no case has the second eye been subsequently involved."

Weymann of Los Angeles replied: "Today I would feel that permanent damage to the optic nerve is not accomplished so quickly as one might think, so that in the absence of evidence of disease of the nervous system, specific disease, or other frank focal infections, I should still be inclined to wait a couple of weeks before opening up the sinuses if there was no great evidence of involvement; and then only with the idea of producing a reaction in the region of the optic nerve which might lead to its recovery, rather than by the elimination of infection present in the sinus."

Lancaster of Boston reported no recent cures by operation on the sinuses but agreed that where all other neurologic evidence is negative an operation on the sinuses might benefit because of the hyperemia which it produces.

Friedenwald of Baltimore replied in part as follows: "I do not feel that in the great bulk of cases the relation of sinus disease to

retrobulbar neuritis has been established. However, I have seen two cases in which I felt sure the sinus infection was definitely related to the ocular symptoms. Both of these patients had slight exophthalmos, pain on pressure over the globe, pain on ocular movement, and a purulent infection of the ethmoid and sphenoid sinuses on the side of the ocular trouble, and both recovered promptly after drainage of the sinuses."

There were, however, two reports from leading ophthalmologists that tended to support the ideas of the responding rhinologists.

Berens of New York said: "I have found a number of cases improved or cured by operation, but I do not advise surgery until local nasal treatment and the use of specific and nonspecific antigens for varying periods have been unsuccessful."

Peter of Philadelphia was very enthusiastic in his support of operation and I quote his reply at length: "I find a very large number of cases of retrobulbar neuritis due to sinus disease. In some instances the cure can be established by medical means, but in the majority of instances the retrobulbar neuritis is converted into frank optic neuritis and papillitis, and in some instances a papilledema, if operative interference is not practiced.

"Such papers as were presented from the Mayo Clinic are very misleading. Either these men see none of the acute cases or their observations have not been very accurate. I have thought it was the former reason.

"The subject is very timely and a most important one to bring before the profession, because it will give an opportunity of stressing the sinuses as the most outstanding factor."

The reports from rhinologists were more in accord.

Beck of Chicago answered as follows: "Merely from memory I recall seventeen cases of marked loss of vision, usually more in one eye than in the other, in which ophthalmologic as well as the rhinologic findings were practically negative. However, the blind spot was enlarged in most of them. I have observed three of these cases long enough now to state positively that they were progressive multiple sclerosis. Two others were alcohol-tobacco amblyopia."

Fenton of Portland, Ore., replied that he could recall offhand several cases which had been cured by operation. He feels that where rapid loss of vision is taking place the patient should receive the benefit of the doubt and decongestive measures involved in improved posterior sinus drainage should be made use of.

Hurd of New York has had very brilliant results but there was evidence of sinus trouble with good x-ray films.

Ridpath of Philadelphia: "In spite of the diversity of opinion between the ophthalmologists and the sinuologists, it is my opinion that infections of the postethmoid and sphenoid cells can be, and are, a decided factor in retrobulbar neuritis."

In 1932 I reported before the New Jersey State Medical Society two cases of sinus origin, in both of which I obtained almost complete cure by operation.

How can we settle this question? In fact, how can any disputed question in medicine be settled? Apparently it will always be one of the medical riddles, and each rhinologist will be forced to use his best judgment in deciding what procedure to follow.

There does seem to be proof that the optic nerve can be affected to a mild degree in quite a percentage of cases. Then why not in the occasional case in which we are particularly interested?

With the present knowledge of sinus anatomy and operative dexterity that most well equipped rhinologists possess, operation on the sphenoid and ethmoid sinuses should not be such a dangerous procedure. An occasional needless operation on a case later proven to be multiple sclerosis cannot begin to hold for us the chagrin that we would feel over a blind patient whose sight might have been saved had we been bold enough to insist on operation at the time when sight might have been restored.

501 GRAND AVE.

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Clinical Notes and New Instruments.

XXVIII.

EXOPHTHALMOS OF OBSCURE NASAL ORIGIN: REPORT OF SIX CASES.*

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While the classical picture of cellulitis or abscess of the orbit accompanied by frank suppuration in a neighboring sinus presents very little difficulty in diagnosis, there are times when profound disturbances in these tissues are caused by a sinus disorder which produces such meager symptoms and signs that its presence and significance may very readily be overlooked. One is impressed by the fact that vascular changes in the orbit, at times reaching the extent of a marked exophthalmos with complete fixation of the globe, may often be brought about by a seemingly mild and insignificant sinus disease.

The extremely grave nature of orbital cellulitis makes it imperative that the cause of the edema be recognized early so that remedial measures, whether by local treatment or surgery, may be instituted without loss of valuable time; for it is only by this prompt action that many cases of orbital abscess with its attendant dangers are to be aborted.

The following cases, which have come under my observation during the past few years, illustrate some of the difficulties in diagnosis encountered in this condition and have been selected because of the fact that in them orbital changes were produced, the extent and gravity of which were out of all proportion to the seemingly mild and inconsequential nasal pathology.

No cases of orbital infection caused by frank suppuration in the sinuses have been included in the following report, but only those in which there existed a certain amount of doubt as to their nasal origin.

*Read before the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Norfolk, Va., January 19, 1935.

REPORTS OF CASES.

CASE 1.—White, male, 3 years of age, admitted to hospital March 16, 1928, with following history. Two weeks ago he had a severe cold with copious nasal discharge which lasted one week. Two days after all symptoms had subsided there appeared a watery discharge from the right eye. This increased in amount and swelling of the lids appeared during the next forty-eight hours. He was put to bed and local treatment to the eye and nose instituted. In spite of this he became progressively worse until, at the time of admission, the eye presented an alarming picture. There was marked swelling of both lids with chemosis of the bulbar conjunctiva, the globe was protruding symmetrically with complete fixation, temperature 101 degrees, leucocyte count 18,600, urine negative. Pain was not a prominent symptom, nor was there evidence of great prostration. Examination of the nose showed moderately swollen turbinates on both sides which were readily shrunk down with a weak ephedrine and cocain solution. No nasal nor postnasal discharge was evident. X-ray examination showed a decided clouding of the antrum and anterior ethmoids on the right side.

Since appropriate local treatment had been carried out for several days without effect, the anterior ethmoids and antrum on the right side were opened intranasally under general anesthesia. The following day, after the removal of the packing, a sanguineous discharge appeared from the right nostril which was so profuse as to necessitate the constant cleansing of the lip. This was accompanied by a very noticeable amelioration of the orbital swelling and exophthalmos until, by the fifth day, the globe had assumed its normal position, and free movement in all directions was restored. In the meantime the nasal discharge had become quite thick, and at the end of ten days had entirely ceased.

During the six years subsequent to this attack he has had occasional severe colds without any recurrence of the orbital inflammation.

CASE 2.—White, male, 8 years of age, was examined in the home February 8, 1930. He had had a mild attack of grippe which was accompanied by a moderate amount of nasal discharge. For the past two or three days he had apparently been in perfect health with no discharge present. During the past twenty-four hours he had complained a great deal of headache, pain in the left eye, and during the night the eye had swollen to such an extent that the lids could not be voluntarily moved.

At the time of examination the temperature was 101 degrees, there was marked swelling of both lids and conjunctiva, and the eyeball was pushed forward and toward the temporal region. No intra-ocular examination was made. The nasal mucosa was moderately inflamed with a very definite swelling of the middle and inferior turbinates on the left side. After thorough shrinkage some mucopurulent material was observed in the left middle meatus, and irrigation with saline solution produced a small quantity of mucopus. Transillumination was clear on the right side, but the left antrum was hazy. Nasal treatment, consisting of shrinkage and irrigation, was carried out twice daily and by the third day the eye had regained its normal appearance and function.

These two cases were undoubtedly early orbital cellulitis, and most certainly would have gone on to abscess formation had effective nasal treatment not been started promptly.

CASE 3.—White, female, 35 years of age, first seen March 9, 1930, at which time she had a peritonsillar abscess on the right side. This was opened and drained, and a normal convalescence ensued. A few days after recovery from the abscess she began to complain of a severe pain in and around both eyes, more particularly on the right side.

Nasal examination in the home revealed no abnormality. The turbinates were normal in appearance, there was no discharge present, and transillumination was exceptionally clear.

Twenty-four hours later the pain had increased and was accompanied by some photophobia and a noticeable swelling of the lids on the right side. After removal to the hospital, general examination, including neurologic, was essentially negative, temperature 99.4 degrees, pulse 88, respiration 20, leucocyte count 17,200. Examination of the nose showed no signs of acute inflammation. The turbinates were normal in size and appearance, and the most searching and careful inspection revealed no mucopurulent material in the nose or nasopharynx. The olfactory fissure was clear, but a probe could not be passed into the sphenoid ostium. X-ray examination showed the sinuses to be entirely negative. By the following morning the condition had progressed and the left eye had also become involved. At this time an ophthalmologic examination showed a marked exophthalmos of both eyes with almost complete fixation of the globes. There was a decided photophobia and a moderate amount of edema of the lids. The bulbar conjunctiva was quite edematous and injected, the cornea appeared normal, the iris clear, and the pupils contracted. Tearing was fairly profuse, but no mucopurulent discharge was present.

Since she had had so recent a peritonsillar abscess, and since the nose seemed so unusually clear, we felt that we must be dealing with a cavernous sinus thrombosis, although no other signs than those mentioned could be made out. The following day the situation became rapidly worse. The edematous conjunctiva protruded from between the lids on both sides, the lids themselves were markedly edematous and injected, the globes were fixed, and pain was intense. The cornea was watched for any pressure changes and an external canthotomy considered. The fundi showed deeply congested veins almost black in appearance. No other changes were present. Twenty-four hour blood culture was negative, and her temperature had not gone above 99 degrees. In the meantime the search for nasal infection was continued. Twice daily the turbinates were shrunk down, suction applied, and the nose irrigated with saline solution. On the third day after admission our persistence was rewarded. While all previous irrigations had returned unusually clear without even a shred of mucus, on this day a large well-formed globe of clear mucus about 20 mm. in diameter came away in the solution, followed by several smaller pieces.

During the next twelve hours the change in her condition was truly dramatic. The pain was almost immediately relieved, the swelling of the lids and exophthalmos rapidly receded, so that by the following day there was a very noticeable improvement in the ocular symptoms. At each subsequent nasal treatment for several days a fair amount of mucopus was obtained from the postethmoid sphenoid region on both sides. The conjunctiva receded behind the lids and ocular movements were so rapidly restored that by the third day following the first evacuation of sinus discharge, free motion was established in all directions and the globe had assumed its normal position. After remaining in the hospital three more days

for nasal treatment and observation, she was discharged symptom free. Some months later ophthalmologic examination showed 20/15 vision in each eye and since that time there has been no recurrence of nasal or ocular symptoms.

The very sudden and complete restoration of function immediately following the appearance of nasal discharge and the afebrile course of the disease led us to the conclusion that the startling changes in the orbit had been caused more by the blocking of orbital circulation from a practically symptomless sphenoid infection than from an orbital cellulitis caused by direct extension of infection.

CASE 4.—White, male, age 35. First examined in the office August 14, 1930. During the past six months he had had two severe colds during both of which there had been some inflammation of the eyes. About ten days ago he had another severe cold with a large amount of nasal congestion and discharge. The discharge had practically ceased, but for about two or three days there had been some pain around the left eye. When he awakened this morning there was a marked swelling of the lids on the left side with profuse tearing. Examination by his ophthalmologist showed the bulbar conjunctiva to be very edematous; there was limitation of motion accompanied by a very definite protrusion of the globe outward and toward the temporal region, the pupil was dilated, and the retinal veins dark and full.

Examination of the nose revealed a very decided acute inflammation and swelling of the middle turbinates on both sides. Thorough shrinking and irrigation produced a large amount of mucopurulent material which seemed to come from the middle and superior meatuses on both sides. Transillumination was clear over the frontal regions, but both antra were somewhat hazy. X-ray examination showed only a slight clouding in the left frontal.

Vigorous local treatment was carried out twice daily for three days, at the end of which time, the swelling and exophthalmos had entirely disappeared. At the end of one week the sinuses appeared entirely clear.

The interesting feature of this case is that, during the four years since the original attack, this same patient has had the same condition in either one or both eyes at seven different times. The left side has been more frequently involved than the right. On each occasion, it has responded very quickly to local treatment and in most instances he has pursued his work with practically no interruption during the course of the attacks.

It would seem quite probable that in this case there is a bony dehiscence which facilitates the transmission of nasal inflammation to the orbital tissues, but he has refused any surgical procedure for permanent relief of the condition.

CASE 5.—White, female, 60 years of age, examined in my office December 3, 1931. Two weeks ago she had noticed some swelling in the left eye. Ophthalmologic examination showed a moderate edema of the lids with widening of the palpebral fissure. Exophthalmometer reading, right eye 18, left eye 24. The fundus was normal. This condition remained present with slight variations for a week, at which time the exophthalmometer reading was right eye 22, left eye 30. There was no history of any recent cold. She had not noticed any nasal obstruction, nasal or postnasal discharge. The only symptom elicited was that of a mild sore throat which she had had previous to the attack.

Nasal examination showed a very narrow nose with both middle turbinates impinged on the septum. There seemed to be a distinct inflammation and swelling of both middle turbinates. After thorough shrinking, a probe could be passed

with difficulty between the middle turbinate and septum on the left side, but the sphenoid ostium could not be located. X-ray examination showed a very slight thickening of the lining mucosa of both antra. The nose was packed with argyrol tampons, after which suction was applied and the nose irrigated with saline solution. A few pieces of mucus were obtained. The following day she felt that there had been a decided improvement in the eye. The nose was treated daily for several days and on each occasion some mucus was washed out. In the meantime she had been getting a very active postnasal discharge and at the end of the fifth day all swelling had disappeared. The exophthalmometer reading was right eye 21, left eye 22. She was then treated at less frequent intervals until the postnasal discharge had entirely cleared.

During the three years since the original attack she has been seen two or three times each year with an acute rhinitis. Only on one occasion has there been any recurrence of the orbital symptoms. This consisted of a very slight swelling of the lids with no exophthalmos and cleared up within forty-eight hours under treatment.

CASE 6.—White, female, age 38, first seen November 21, 1933. Four days before she had noticed some swelling around both eyes, more particularly the left, until at the time of examination there was a marked edema of the conjunctiva present. She complained of no pain except when the eyes were rotated. There was a very decided proptosis slightly toward the temporal region, and a very definite diplopia in the upper field. Vision 20/15 in each eye. Fundi appeared normal. Movements of the left globe were limited in all directions. Examination of the nose showed a moderate amount of congestion in both middle turbinates. After thorough shrinkage, no discharge could be made out. X-ray examination at this time showed clouding over both antra and both anterior ethmoids, but the involvement seemed to be more on the right than the left. The frontals and posterior sinuses were clear. Transillumination showed slight haziness over both antra. The left antrum was punctured and irrigated, the solution returning practically clear.

She was sent to the hospital for nasal treatment which consisted of thorough shrinking, argyrol packs, and irrigation. For forty-eight hours there was no improvement in the condition, and we were on the point of advising an intranasal ethmoidectomy and antral window when some mucopurulent material began to be discharged. There was immediate improvement in the orbital condition and in about four days the eye had attained its normal position with restoration of free movement. The sinus condition was rather obstinate and persisted for several weeks. Since that time she has had two or three colds but has had no recurrence of the sinus or orbital involvement.

SUMMARY AND CONCLUSIONS.

It would seem that from consideration of this admittedly small group of cases a few interesting observations may be made:

1. In any case of orbital edema careful and repeated examinations of the sinuses should be made and every resource at our command used before concluding that the sinuses are not the causative factor. Perseverance may bring to light an obscure and apparently insignificant infection.

2. There may be a complete absence of any history of recent nasal inflammation, the condition being so slight as to entirely escape the notice of even an intelligent patient.

3. Obscure lesions in the postethmoid sphenoid region may produce circulatory disturbances in the orbit which closely resemble cavernous sinus thrombosis.

4. Because in each instance improvement became apparent within a very short time after the institution of effective nasal treatment, it seems certain that, if seen and diagnosed before the formation of an orbital abscess, a large proportion of these cases may be permanently relieved by local treatment or minor intranasal procedures.

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XXIX.

A BRONCHOSCOPIC AND ESOPHAGOSCOPIC TELE-MAGNIFIER.*

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During a bronchoscopic, esophagoscopic or direct laryngeal examination, it is frequently desirable and most informative to be able to view the lesion or, in the presence of a foreign body, to study the mechanical problem involved, under magnification, comparable in some respects to the advantages and information to be gained when examining the ear with an otoscope.

Several means for obtaining an enlarged or magnified view have been devised, some with multiple eye pieces for teaching purposes. These accessories have the objection that they either do not offer sufficient magnification or do not permit of operative procedures being carried out while the field is under magnification, and serve only as accessories for examination.

For the past three years experimental work has been carried on, resulting in the development of a tele-magnifier, which gives a magnified image of approximately three and one-half times. The tele-magnifier is attached to the proximal end of the bronchoscope or esophagoscope by means of two small posts and extension arms. Through a hinged joint on the extension arms the tele-magnifier can be tilted up and out of the field of the parallel axis of the bronchoscopic or esophagoscopic tube as desired. Thus, instrumentation into and through the tubes can be carried out with the tele-magnifier in place or tilted up, as the operator chooses.

The tele-magnifier can likewise be quickly attached or detached, and can be focused for different distances or for use with different length tubes.

There is no obstruction to the patient's breathing when the tele-magnifier is in place or attached. It is light in weight and offers no hindrance in any way to the operator or his assistants.

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*Presented before the American Laryngology, Rhinology and Otolaryngology Society, June, 1933, Chicago, Ill.

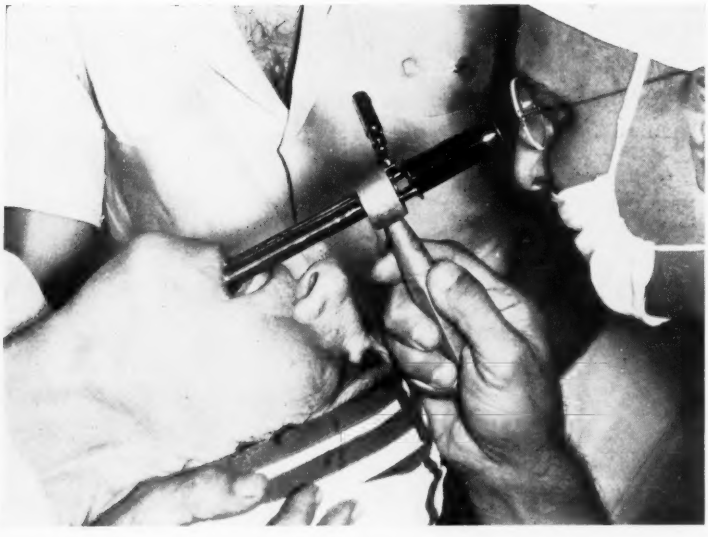


Fig. 1. Showing diagnostic tele-magnifier attached to the tube my means of two short posts. The tele-magnifier can be swung out of view at a moment's notice and still remain attached to the proximal end of the tube.

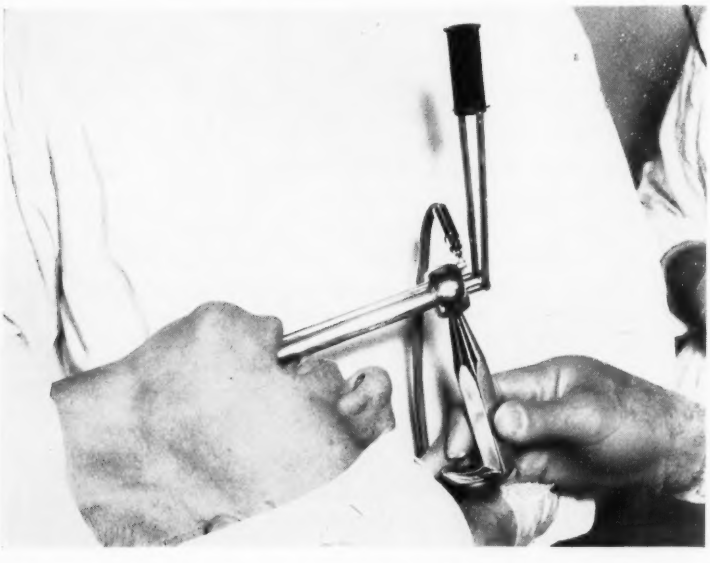


Fig. 2. Showing tele-magnifier tilted up and out of the field of view through the tube.

Fig. 3. Showing tele-magnifier with extension arms attached to bronchoscopic tube. Forceps introduced through the tube under magnification in order more easily to carry out the necessary operative procedure.

The fact that one is able with the tele-magnifier to get a highly magnified view of the lesion or object, in the field of view at the distal end of the tube, and to study the pathology or mechanical problem present and execute the surgical necessities arising, is a distinct advantage when working in the esophagus or tracheobronchial tree.

The tele-magnifier is available with or without the extensions, depending on whether or not it is to be used for diagnosis only or for surgery.

I am indebted to the Electro-Surgical Instrument Company, of Rochester, N. Y., for executing the mechanical details in the development of the idea.

2010 Esperson Bldg.

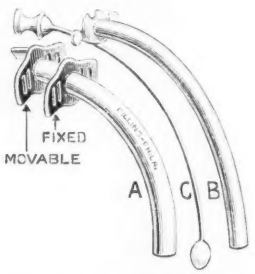
XXX.

A MODIFIED TRACHEOTOMY TUBE.

LE ROY A. SCHALL, M. D.,

BOSTON.

Following tracheotomy, there is a variable period during which the tracheal secretion is very profuse. During this time the dressing becomes soaked with secretion and requires frequent changing, necessitating almost constant nursing care. In changing the dressing, the tracheotomy tube is frequently moved, which causes spasms of cough with consequent expulsion of more mucus.



Modified Tracheotomy Tube.

To overcome this difficulty, this tracheotomy tube was devised. It is essentially an extended Jackson tube with a double neck plate; the inner fixed, the outer movable. The tube is inserted in the usual manner, but the extension permits more gauze to be applied without disturbing the curve or fit of the tube.

By its use the tracheal wound is kept drier, therefore cleaner; the patient is more comfortable as he is disturbed less; and less nursing care is required.

When the tracheal secretions cease, the tube may be replaced by any of the other types of tubes which the case may indicate.

270 COMMONWEALTH AVE.

HARMON SMITH, A. B., M. D.

1872-1934.

Doctor Harmon Smith, A. B., M. D., of New York City, was born at McDonough, Georgia, on March 20, 1872, and died suddenly in New York City on December 11, 1934. Doctor Smith was a prominent otolaryngologist and held many positions of honor in the various special societies. He was, perhaps, best known as joint author of a text on Diseases of the Nose and Throat, in collaboration with the late Jonathan Wright.

Doctor Smith was graduated from the University of Georgia in 1892 and from the Bellevue Hospital Medical College of New York in 1897. The honorary degree of D. Sc. was conferred upon him by the University of Georgia in 1922.

He was a Fellow of the American Medical Association and its local component organizations and of the American College of Surgeons. In 1921 he was Vice President of the American Laryngological, Rhinological and Otological Society and President of the American Laryngological Association in the same year. For some years he was professor of laryngology at Cornell University Medical College. At the time of his death he was a director in the throat department of the Manhattan Eye, Ear and Throat Hospital, consulting laryngologist to the Memorial and Babies' Hospitals and consulting aurist to the Monmouth Memorial Hospital, Long Branch.

He is survived by one son, Harmon, aged fifteen.

Abstracts of Current Articles.

NOSE.

Physiologic Researches of the Accessory Nasal Cavities. Third Note: The Sinuses and the Temperature of the Inspired Air (Alcune ricerche intorno alla fisiologia delle cavita accessorie del naso. Nota III: I seni e la temperatura dell'aria di inspirazione).

Bronzini, A. (Pisa), Valsalva, 10:34-39 (Jan.), 1934.

The writer has reported, in 1927 and 1928, other experimental results on the physiology of the nasal accessory sinuses. He now reports the latest results obtained from his experiments on dogs, concluding as follows:

In a broad sense, the nasal accessory sinuses are to be considered as organs which serve to warm the inspired air. In this function they do not participate in an active manner, by warming the contained air through the blood vessels of the mucous membrane which lines these cavities, as it is today generally believed.

Bronzini contends that to this function the sinuses are to be considered as reservoirs for warm air. The warm air enters the sinuses at the very beginning of the expiration, and empties them during inspiration. This air which fills up the sinuses is warm, humidified and oxygenated because it does not come in contact with the alveolar tissue but remains in the uppermost portion of the respiratory tract.

The difference in temperature between the air contained within the sinuses and the external air is higher when the external air is colder. These cavities constitute a true and proper apparatus of compensation, which modifies the temperature of the inspired air in a manner relatively proportionate to the requirements of the organism.

SCIARRETTA.

The Olfactory Function in the Newborn (Sulla funzione olfattoria nel neonato).

Ciurlo, L. (Milano), Valsalva, 10:22-33 (Jan.), 1934.

Ciurlo reviews the literature thoroughly and reports his experiment in detail. He was able to demonstrate that a newborn is able to perceive odors from the very first hours of his birth, and probably an odorous substance is capable of producing its particular olfactory sensations in these infants.

SCIARRETTA.

Incidence of Allergy in Rhinologic Practice.

Baum, Harry L., M. D. (Denver), Arch. of Otolaryng., 20:804 (Dec.), 1934.

In the normal course of rhinologic practice approximately 27.3 per cent of all patients with nasal complaints may be expected to exhibit some form of allergy in the nose, and of these, about 89 per cent will show allergic changes in the sinus mucosa.

A better understanding of allergic nasal and sinus disease on the part of rhinologists will prevent frequent errors in the diagnosis of suspected focal infections.

A realization of the true relation of nasal and sinus allergy to other allergic manifestations throughout the body will enable the rhinologist to avoid many useless and damaging operations, with destruction of innocent and valuable tissue under the mistaken assumption that dangerous foci of infection are being removed.

TOBEY.

Ozena and Parathyroid (Ozena e paratiroidi).

Briani, A. A. (Rome), Valsalva, 10:1-22 (Jan.), 1934.

The author discusses the numerous therapeutic methods employed for the treatment of ozena. He confirms what is today approved by the majority of researchers; that is, the primary and necessary condition for the development of ozena is the constitutional imbalance of the endocrine system. This hormone disturbance will not only cause atrophy of the nasal bones and mucous membrane, but also a reduction of the body resistance, giving the bacteria a rich medium for their growth, thus completing the typical picture of this disease.

It has been proven clinically and experimentally that hypoparathyroidism will produce the following: hypocalcemia, decrease of neuromuscular reaction when stimulated by the galvanic current, bodily thermic variations and skeletal changes.

Briani treated four typical ozena cases with daily intramuscular injections of large doses of active parathyroid hormone, which was prepared and measured according to Collip's method. In addition to the above, calcium was also given. He reports an immediate improvement in the fetor, which subsequently disappeared; a gradual decrease of scab formation; reappearance of turgescence and moisture of the nasal mucous membrane; return of blood calcium content to normal, and a great improvement of the general physical condition.

The writer also reports a case of chronic atrophic laryngitis with aphonia and the formation of scabs, the former of three months'

duration. Many forms of therapy were used without improvement, but a quick cure was obtained by the use of the parathyroid hormone.

In conclusion, he thinks that ozena may be in relation with the functional deficiency of the parathyroids, which are in a state of miopragia due to congenital or acquired diseases (lues or tuberculosis), and the ozena symptoms appear the instant these glands are called upon to perform more work (calcification of the bones and puberty).

SCIARRETTA.

Solitary Nasal Polyps (Les polypes solitaires des fosses nasales).

Alonso, Prof. J. M., and Pietra, Dr. P. (Montevideo). Rev. de Lar., Otol., Rhin., 55:1069 (Nov.), 1934.

Careful study of eleven cases, illustrated by excellent x-rays and diagrams, justify the conclusion that nearly all such polyps come from the maxillary mucosa. Diagnostic puncture with use of lipiodol is recommended. The pedicle is most frequently found in the alveolar recess, but may come from the roof or outer wall. Removal without recurrence requires radical operation through the canine fossa.

FENTON.

Duplex Maxillary Sinuses (Seios maxilares duplos).

Leite, Dr. A. O. (Ris Frande do Sul), Rev. Oto. Lar. S. Paulo, 2:393 (Sept.-Oct.), 1934.

This is a case report of bilateral accessory maxillary sinuses, lying in the mesial aspect just above the lateral incisors on both sides, and probably of dental origin, since one was filled with foul pus from a dental root, and neither had any nasal ostium.

FENTON.

PHARYNX.

Perilaryngeal and Pharyngeal Suppurations (Les suppurations périlaryngo-pharyngées).

Truffert (Paris) and Viela (Toulouse). Report to 47th Congress of O. R. L., Paris, 1934. O. R. L. Internat., 18:677 (Oct.), 1934.

All suppurations near the posterior pillar of the fauces or the floor of the mouth tend to point toward the hyo-thyro-epiglottic space, between the thyrohyoid membrane and the anterior face of the epiglottis. Acute abscesses in this region may require opening below the hyoid, unless pointing upward occurs early. Diffuse forms may become extremely grave, with extreme dysphagia and a croaking voice.

Laryngeal abscess is primarily influenzal or traumatic; secondarily, from extension of neighboring suppuration or secondary infection of a specific perichondritis. It is most frequent in the epiglottis, next the arytenoids, secondarily the posterior wall of the larynx or the pharyngolaryngeal gutter. Tuberculosis, syphilis, cancer or necrosis from physical agents used in treatment (radium) are blamed for perichondritis with abscess.

Retrolaryngeal and pharyngeal abscesses, when acute, are due to glandular infection or to trauma of the pharyngeal musculature and mucosa. If a good-sized collection of pus can be localized internally, it should be widely opened within (having in mind the danger of severe delayed hemorrhage.—Abstractor); if this is not possible, and x-rays show definite localization, lateral cervical drainage must be made. Cold abscesses of this region are always tuberculous, arising from the neighboring vertebræ, and require lateral opening, with orthopedic immobilization of the cervical spine.

FENTON.

Systemic Researches on the Presence of Osseous and Cartilaginous Tissue in the Palatine Tonsils (Ricerche sistematiche sopra la presenza di tessuto osseo e cartilagineo nella tonsilla palatina).

Pellegrini, G. (Milano), Arch. Ital. di Otol., Rino. and Laring., 46:180-203 (March), 1934.

This article contains a good review of the literature. The conclusions drawn are from a meticulous histologic examination of 432 tonsils.

1. The cartilaginous and osseous tissue was found in the palatine tonsils very often (25.46 per cent of the examined tonsils).

2. These types of tissues are present more frequently in older individuals.

3. In the newborn and children, the cartilage must be considered of embryonic origin, probably from wandering germs of the second branchial arch.

4. The cartilaginous inclusion found in the adult individuals originated from metaplasia of the connective tissue as a result of chronic inflammatory stimulation.

5. The osseous tissue is never found in tonsils of individuals under the age of 15 years. Being of inflammatory origin, it may take its origin directly from connective or cartilaginous tissue.

Seventeen interesting photomicrographs accompany the article.

SCIARRETTA.

LARYNX.

Vicarious Vocal Mechanisms.

Kallen, Leo A., M. D. (New York). Arch. of Otolaryng., 20:460 (Oct.), 1934.

The author believes that operative methods of removing the larynx have made greater progress than methods of restoring a voice to larynx-less people, and this article deals with the history and chronology of vocal aids, the disadvantages of present laryngeal prostheses and urges the development of a vicarious voice. Early cases of pharyngeal voice are cited. The physiology of voice and speech production is explained and the vicarious air chambers and vicarious larynx are carefully described.

The vocal prognosis in laryngectomized patients is more favorable than in any other field of vocal pathology, and only mental depression can seriously interfere with progress.

TOBEY.

EAR.

Contribution on Etiological Investigation of Otosclerosis (Contributo alle conoscenze sulla etiologia dell'otosclerosi).

Torrini, G., Jr. (Firenze), Valsalva, 10:347-357 (May), 1934.

The article contains a precise review of the histologic changes found in the bony capsule of the otosclerotic ear. There is also a comprehensive summary of the outstanding theories advanced on the etiology of otosclerosis.

Torrini's experiments are based on the hypothesis of those scientists who consider the pathogenesis of otosclerosis to be the results of endocrine dysfunction.

He selected a number of male and female guinea pigs, their age approximating the period in which man develops otosclerosis. A couple were retained for control. In a group of guinea pigs, one ovary or testicle was removed. In another group, a bilateral orchidectomy or oöphorectomy was performed. At the appropriate time all animals were sacrificed, and immediately the two petrosal bones were removed from each animal.

These temporal bones were properly prepared and sectioned. The histologic study of these sections did not show the slightest change as compared to the normal.

He concludes that partial or total gonad's dysfunction is not the only etiologic factor, if in any way at all responsible for the cause of otosclerosis.

SCIARRETTA.

On the Function of the Saccule.

Ashcroft, D. W., and Hallpike, C. S. (London), J. of Laryng. and Otol., 49:450 (July), 1934.

The authors review the history of our knowledge of the physiology of the saccule and distinguish four well marked periods, beginning with the work of Mach, Breuer and Crum Brown, which established the endolymph movement principle of canal function, down to the time of Versteegh and other writers investigating the theories of Magnus.

In the experimental work described by the authors, the problem of saccular function was approached upon electrophysiologic lines, the animal employed being the frog. With a micromanipulative technic in combination with a conventional amplifying system and a cathode ray oscillograph, action currents were recorded in the saccular nerve of the frog. The action currents occur in response to vibrational stimuli, the frequency of the stimulus being reproduced accurately up to a frequency of at least 500. Reasons are given for regarding the electrical charges recorded as true action potentials.

It is concluded that the saccule in the frog is an organ of vibration sense. The authors believe that the experimental results support the suggestion of Tait that in man the saccule is concerned in the reception of bone conducted sound.

GOLDSMITH.

Prenatal Medication as a Possible Etiologic Factor of Deafness in the Newborn.

Taylor, H. Marshall, M. D. (Jacksonville), Arch. of Otolaryng., 20:790 (Dec.), 1934.

Certain drugs have a predilection for the auditory nerve. Idiosyncrasy for drugs may be an important factor in causing nerve deafness.

Quinin takes precedence among the drugs causing nerve deafness and is frequently used during the term of pregnancy.

Evidence has been presented that certain drugs which have a predilection for the auditory nerve, when administered to the pregnant mother, readily pass through the placenta and may be toxic to the fetus, a possibility which the otologist has virtually ignored.

Prenatal medication as a possible etiologic factor of deafness in the newborn is of sufficient importance to warrant the co-operative research of the biochemist, the histopathologist, the obstetrician and the otologist.

TOBEY.

Industrial Accidents to the Ear (Oreille et accidents du travail).

Causse (Paris). Report to the 47th Congress of O. R. L., Paris, 1934. O. R. L. Internat. 18:681 (Oct.), 1934.

Only Germany, Bulgaria, Mexico, the Soviet Republics, and Czechoslovakia recognize industrial deafness in their labor laws.

Most accidental damage to the ears is traumatic. Longitudinal fractures open the middle ear, respect the internal ear and run along the anterior face of the petrous. Transverse fractures gravely injure the internal ear but respect the middle ear. The major portion of these injuries is found among victims of concussion, where there is no relation between the intensity of the trauma and the importance of its sequelæ. Sometimes deafened, such cases are almost always afflicted by vertigo.

Accidents from electricity are rare, resulting in annoying later cochleovestibular symptoms, whether from deep burns or from lightning. Compressed air workers are especially liable to gas emboli of the cochleovestibular apparatus, causing very sudden and severe vertigo and/or deafness shortly after or during return to normal air pressure.

Intoxications are infrequent, either irritative or producing certain reductions in hearing, and are due to lead, mercury, arsenic, phosphorus, and to some extent carbon monoxide.

Prophylaxis of occupational deafness by noise abatement, and the study of methods for its relief, are of interest not only to the worker and the otologist but to the public authorities. Detection of simulation does not require elaborate equipment.

FENTON.

Vitamines in the Treatment of Otosclerosis (L'importance des vitamines dans la thérapeutique de l'otosclérose).

Kaban, D. S. (Kiev), Rev. de Lar., Otol., Rhin., 55:1171 (Nov.), 1934.

Finding nothing unusual in the blood calcium and about 15 per cent of eosinophilia, the following group treatments were used: (a) feeding dry yeast, no results after three months; (b) administration of cod liver oil, no results as to hearing, improved general physique; (c) feeding dry yeast plus skin irradiation by the quartz lamp —of 17 cases, 9 reported 60 to 70 per cent improvement in subjective noises, improved hearing and general physique, results were not so good in six others and there was no change in two; (d) feeding of yeast subjected to quartz irradiation: rapid improvement, lasting, however, only ten to fourteen days; (e) feeding of irradiated yeast

plus the congestive effect of Stcherbacov's "mud collar" every other day, six to twelve times: Four cases much helped after six weeks, the other eight having a return of tinnitus and hearing again reduced but not so far as at first; (e) combined treatment, plain yeast, "mud collar" and quartz irradiation of the whole body: of sixteen treated, ten reported freedom from tinnitus and much improved hearing one month after cessation of treatment, five were helped considerably and one required other (general) treatment.

The author requests that others undertake a check of his methods, in the hope that fixation of vitamin D in the skin, and improved internal ear circulation may be thoroughly tested.

FENTON.

Treatment of Labyrinthitis.

Friesner, Isidor, and Rosenwasser, Harry (New York), Arch. of Otolaryng., 20: 139 (Aug.), 1934.

The authors review the opinions of various European and American authorities and give their own ideas of the proper treatment for this disease. Several classifications of labyrinthitis are mentioned. The conservative and radical procedures for circumscribed, latent and manifest labyrinthine disease are discussed. Only in the circumscribed is complete accord found.

TOBEY.

Bacteriology of Acute Infections of the Middle Ear and Mastoid.

Page, John R., M. D. (New York), Arch. of Otolaryng., 20:447 (Oct.), 1934.

Three hundred cases were studied in this series; 231 cases presented unperforated ear drums needing myringotomy, while sixty-nine cases had ears that were discharging. The method of obtaining the cultures is described in detail. Four cultures were taken from the middle ear and one from the eustachian tube. Seventeen per cent, or 51 patients, had infections due to pneumococcus type III; thirty-five were followed, and twenty-nine recovered without operation for mastoid. Pneumococcus type III was not found to be as insidious as other investigators have experienced it. Ninety-two, or 30.6 per cent, were due to hemolytic streptococcus infection. Fifty-one were followed and nine, or 17.6 per cent, had operations on the mastoid, while 42, or 83.4 per cent, recovered without operations. Operation on the mastoid was performed three times as frequently on patients whose ear drums had ruptured spontaneously as on those having early paracentesis.

TOBEY.

Tuberculoma of the Brain Associated With Ear Disease.

Stewart, J. P. (Edinburgh), J. of Laryng. and Otol., 49:493 (Aug.), 1934.

The author reviews the literature on tuberculosis of the brain and reports a case of a tuberculoma complicating a tuberculous mastoiditis. The route of the infection was traced from the primary focus—tuberculosis of the adenoid tissue, eustachian tube, middle ear, mastoid—and finally to the brain tissue. Microphotographs of these tissues are presented. A direct spread was demonstrated and not the usual spread of the infection by way of the blood stream.

Diagnosis in these cases is difficult unless localizing pressure symptoms are present. As no such symptoms existed in the case reported, the diagnosis of tuberculoma was made only at operation. Prognosis is bad, cases rarely living more than one year. Treatment in this case consisted in a decompression, which had already been performed by nature, removal of the diseased bone, the tuberculoma left in situ and ultraviolet radiation. The case reported has to all appearances done very well, although one must be very cautious, as it is only six months since the operation. Extirpation of the tuberculoma is not to be recommended.

GOLDSMITH.

The Relation of the Internal Auditory Artery to Atherosclerosis (L'arteria uditiva interna nei rapporti con l'aterosclerosi).

Bozzi, E. (Milano), Arch. Ital. di Otol., Rino. and Laryng., 46:100-132 (February), 1934.

These interesting pathologic-anatomic studies are reported in a thirty-two page article containing ten beautiful histologic illustrations.

In the first part of this work the writer reviews extensively the varied and doubtful opinions of many researchers as to the relation of presbyacusis to atherosclerosis. Then he gives the detailed anatomy of the internal auditory artery, its division, anastomosis, etc. He then discusses the findings reported by a large number of illustrious workers, in respect to various pathologic changes found in this artery and how these alterations cause disturbances of the peripheral auditory end organs.

Therefore, Bozzi conducted histologic examination of the extraosseous (free) portion of two hundred internal auditory arteries obtained from one hundred autopsies of individuals, mostly over fifty years of age. The purpose of this was to determine if the internal auditory artery is prone to atherosclerotic changes, and if so, the de-

gree and extent of the degeneration, and also if the same pathologic changes demonstrated in the auditory artery would be found throughout the arterial system of the same individual. In 29.7 per cent of these cases atherosclerotic changes were found in the internal auditory artery. The extent and degree of the pathologic variations were divided into three classes. He concludes by giving a statistical scale of atherosclerotic changes found in the arteries of the various organs of the individual demonstrating sclerotic changes of the auditory artery.

SCIARRETTA.

Roentgen Changes in the Petrous Portion of the Temporal Bone Without Clinical Manifestations.

Coates, George M., M. D., Ersner, Matthew S., M. D., and Myers, David, M. D. (Philadelphia), Arch. of Otolaryng., 20:615 (Nov.), 1934.

A review of the literature relative to the involvement of the apex of the petrous portion of the temporal bone has been given.

The relationship of Gradenigo's syndrome to petrositis has been emphasized: (a) Gradenigo's syndrome is independent of petrositis; (b) Petrositis may produce the Gradenigo's syndrome; (c) Petrositis may exist without any symptoms.

It has been stated that Gradenigo's syndrome, or petrositis, may be due to venous congestion of the temporal bone resulting from venous circulatory disturbances.

In many instances the roentgenologist is unable to distinguish between petrositis, exudative petrositis and congestion of the petrous portion of the temporal bone due to venous anomalies of the skull.

According to Wittmaack, Profant, Druss, Kopetzky and others, true petrositis can occur only in a pneumatized temporal bone.

In a diploic temporal bone the lesion is usually osteomyelitic rather than coalescent.

Petrositis, as revealed on roentgenographic examination, does not necessarily indicate surgical intervention.

The Kopetzky-Almour operation or other operations on the petrous portion of the temporal bone are not our first consideration, as many patients recover after simple mastoidectomy and myringotomy.

Key films are essential for the following reasons: (a) to determine the type of temporal bone, whether pneumatic, sclerotic or

diploic; (b) to determine the size of the lateral sinus and jugular foramina, and (c) for the roentgenographic visualization of the temporal bone for a permanent record. Should signs and symptoms appear, the plates become of intrinsic value.

A series of nine cases were presented, each of which showed definite roentgenographic evidence of involvement of the apex of the petrous portion of the temporal bone. Recovery occurred in each of these cases. One patient recovered following a Kopetzky-Almour operation. Another patient recovered as the result of several myringotomies. Rare judgment and care are necessary in deciding on the treatment in each case. Radical surgical procedure should always be based on definite indications.

TOBEY.

MISCELLANEOUS.

Use of Extract of the Suprarenal Cortex in Pyogenic Infections.

Wenner, W. F., and Cone, A. J. (St. Louis), Arch. of Otolaryng., 20:178 (Aug.), 1934.

Five cases are reported in detail and the investigators feel that cortical extract was effective in bringing about improvement in pyogenic infections. It reduces the temperature when elevated and makes the patient feel better and able to undergo operative procedures more satisfactorily. In patients with poor resistance to infection the best results were obtained when the extract was given at forty-eight hour intervals.

TOBEY.

Ethmo-Orbital Osteoma With Pneumo-Encephalocele (Osteoma etmoido-orbitario con pneumo-encefalocele dovuto a prolungamento endocranico).

Malan, A. (Torino), Rit. Oto-Neuro-Oftal. and Radio-Neuro. Chir., 11:47-59 (Jan. and Feb.), 1934.

A report of right ethmo-orbital osteoma observed in a twenty-six-year-old woman. The tumor broke through the orbital roof and the lamina cribrosa, extending into the endocranium. Its extension perforated the dura, making an indentation into the right frontal lobe, thus creating a communication with the anterior pole of the right lateral ventricle.

The patient died thirty-six hours after the operation, from gas embolisms which were found in the various cerebral venous sinuses. There was also a large ventricular pneumatocele.

The author reviews the literature, the mechanism of the air accumulation of the endocranium, the operative indication and the various surgical technics. He discusses Cushing's splendid operative technics and praises the employment of the fascia lata for the closure of the dural wound.

Six illustrations accompany the article.

SCIARRETTA.

Professor Guglielmo Bilancioni, director of the otorhinolaryngologic clinic of the University of Rome, died January 6th, after a year's illness. He succeeded the late Professor Gh. Ferreri at the Policlinic in 1928. He was the founder and editor of "Il Valsalva," and had not only produced over three hundred titles including works of special excellence on ozena and laryngeal tuberculosis, upon developmental pathology in otolaryngology and a treatise on otology, but was an international authority on the history of medicine, especially that of Italy.

FENTON.

NOTICE.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

Examinations will be held in New York City, June 8th, during the meeting of the American Medical Association, and in Cincinnati, in the fall, at the time of the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for application blanks.

W. P. WHERRY, M. D., Secretary-Treasurer.

H. P. MOSHER, M. D., President.

NOTICE.

The Washington University School of Medicine announces the following courses for graduates:

Intensive course in Diseases of the Ear, Nose and Throat, June 24 to July 20.

The Displacement Method of Sinus Diagnosis and Treatment, April 23 to April 26.

Allergy as Related to Otolaryngology, August 12 to August 17.

The Surgical Anatomy of Operative Procedures of Otolaryngology. November 14 to November 18.

Information may be had of the Registrar, Washington University School of Medicine, St. Louis, Mo.

Book Received.

Anatomical Revision of the Arterial System (Revision anatomical del sistema arterial).

Pedro Belou, Professor de la Facultad de Ciencias Médicas de Buenos Aires. Cloth. Octavo of 544 pages with 371 illustrations. Vol I, Technique. Vol. III, Atresias of the Abdomen, Pelvic Cavities and Lower Extremities. Buenos Aires: 1934.

This monumental book is an atlas which represents the work of many years of the author and his assistants at the Department of Anatomy of the Medical School of Buenos Aires. It attempts a revision of the actual knowledge of the arterial system. The author emphasizes that the attempt to add new details to the previous work of several generations of anatomists is well nigh purposeless. To pay a tribute to the men who gave the basis to the present knowledge, he includes at the end of the first volume a most complete list of books and papers dealing with the arterial system, and the methods for its study. The list includes 3500 titles and certainly is the most complete ever published.

In collecting that list the author has rendered an invaluable service to future research. But this is certainly only a small part of the stupendous task he has accomplished.

The first volume of the book is dedicated to the discussion of the technical methods of study of the arterial system. After a summary description of the technique of injection of arteries and capillaries and a very interesting discussion of the photographic reproduction of the anatomic preparations, the author enters into the study of the radiologic technic. The method consists in injecting the arteries with masses opaque to the x-rays and making stereoscopic roentgenograms.

The work of previous investigators is fully considered in the first part of article 4, chapter III, while the rest of the article contains a minute description of the extensive researches of the author with many different masses of injection. He gives preference, for the study of the arterial system down to the precapilliaries, to gelatine masses. One mass is composed of sodium uranate, 20 grams, and gelatine water, 50 grams. The other mass contains a variable proportion of equal parts of mercury iodide, potassium iodide and water, and of

gelatine water. When the capillary net has to be injected soluble masses are necessary.

Chapter IV deals with the technic of stereoradiography as used in his department.

The preparation of the injected specimens for the radiography is minutely described in Chapter V. A radiography of the specimens in toto is often useless because of the complexity of the pictures obtained. As a rule, it is necessary to reduce the size of the preparation, especially those of the head, thus making possible the photograph of only relatively simple territories. The photographs reproduced in this volume are very impressive and clearly demonstrate the usefulness of the radiologic technic for the analysis of the arterial supply of large territories and of small organs. A photograph of the intact specimen is first made and later photographs of sections of it, each time trying to obtain an "optical layer" of the arterial net. Figs. 151 to 167 exemplify the technic followed in the study of the arteries of the head. Fig. 154, with the superficial arteries of the head; Fig. 160, with the meningeal arteries; Fig. 161, with the inferior maxillary artery; Figs. 165-67 with the internal maxillary artery and its branches, and Figs. 171 A and B, with arteries of the ear, deserve special mention.

Volume II is an atlas of the arterial system of the abdomen and lower extremities; it contains photographic reproductions in color of ordinary preparations and roentgenograms. It may be stated that it constitutes the best atlas ever published. That every library should have a copy of it does not need to be mentioned. The publications of the other volumes, with the arterial system of head, neck and torso, must be awaited with interest.

LORENTE DE NO.